CLINICAL PROFILE AND OUTCOME OF PATIENTS PRESENTING WITH SEVERE THROMBOCYTOPENIA IN A TERTIARY CARE HOSPITAL

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Submitted By

DR. MEGHA DADU

DEPARTMENT OF GENERAL MEDICINE
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA, PUNJAB

DECLARATION BY THE CANDIDATE

I hereby declare that the thesis entitled "**CLINICAL PROFILE AND OUTCOME OF PATIENTS PRESENTING WITH SEVERE THROMBOCYTOPENIA IN A TERTIARY CARE HOSPITAL**" submitted towards partial fulfillment of the requirements for the award of **DOCTOR OF MEDICINE** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr. Vikas Garg**, Professor, Department of Medicine, **Dr. Dinesh Gupta**, Professor & Head, Department of Medicine and **Dr**. **Bhavna Malhotra,** Professor, Department of Pathology. No part of this thesis has formed the basis for the award of any degree previously.

_____

Dr. MEGHA DADU
Department of
General Medicine,
Dayanand Medical College & Hospital,
Ludhiana, Punjab

Date:

Place:

CERTIFICATE OF SUPERVISORS

This is to certify that the work incorporated in this thesis entitled "**CLINICAL PROFILE AND OUTCOME OF PATIENTS PRESENTING WITH SEVERE THROMBOCYTOPENIA IN A TERTIARY CARE HOSPITAL"** has been carried out by **Dr. Megha Dadu** under our direct supervision. The data incorporated in this thesis is original and genuine and has been carried out by the candidate herself.

Signature of Supervisor

Dr. Vikas Garg
Professor
Department of Medicine
DMC&H, Ludhiana

Signature of Co-Supervisor

Dr. Dinesh Gupta
Professor & Head
Department of Medicine
DMC&H, Ludhiana

Dr. Bhavna Malhotra
Professor
Department of Pathology
DMC&H, Ludhiana

Date:

ENDORSEMENT BY THE HEAD OF THE INSTITUTE

This is to certify that the thesis entitled "**CLINICAL PROFILE AND OUTCOME OF PATIENTS PRESENTING WITH SEVERE THROMBOCYTOPENIA IN A TERTIARY CARE HOSPITAL "** is a bonafide research work done by **Dr. Megha Dadu** under the supervision of **Dr. Vikas Garg**, Professor, Department of Medicine, **Dr. Dinesh Gupta**, Professor & Head, Department of Medicine and **Dr**. **Bhavna Malhotra,** Professor, Department of Pathology.

Principal

Dr. Sandeep Puri
Principal
DMC&H, Ludhiana

Date:

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Signature

Dr. MEGHA DADU
Department of General Medicine,
Dayanand Medical College & Hospital,
Ludhiana, Punjab

Date:

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Dedicated
to
My Family

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ABBREVIATIONS

APACHE II	Acute physiology and chronic health evaluation ii
APLA	Anti phospholipid antibody
APS	Antiphospholipid syndrome
ATT	Anti-tubercular therapy
CAD	Coronary artery disease
CKD	Chronic kidney disease
CLD	Chronic liver disease
CVA	Cerebrovascular accident
DIC	Disseminated intravascular coagulation
D-ITP	Drug induced thrombocytopenia
DNA	Deoxyribonucleic acid
FDA	Food and drug administration
HCV	Hepatitis c virus
HIT	Heparin induced thrombocytopenia
HLH	Hemophagocytic lymphohistiocytosis
HUS	Hemolytic uremic syndrome
ICH	Intracranial hemorrhage
ICU	Intensive care unit
ITP	Immune thrombocytopenic purpura
IVIG	Intravenous immune globulin
PTP	Post transfusion purpura
RDP	Random donor platelet
SDP	Single donor platelet
SLE	Systemic lupus erythematosus
TPO	Thrombopoeitin
TPO-RA	Thrombopoetin receptor agonist

ABSTRACT

BACKGROUND: Thrombocytopenia is defined as platelet count less than 150 X 10^9 /L. It can further be classified as mild (> 70 X 10^9 /L), moderate (20 - 70 X 10^9 /L) & severe (< 20 X 10^9 /L). The presence of thrombocytopenia in a hemogram should alert the physician to identify the underlying etiology for the prompt management of the patient.

AIM OF STUDY: Analysis to study the clinical profile and outcome of patients presenting with severe thrombocytopenia.

METHODOLOGY: A prospective study was conducted in patients presented with severe thrombocytopenia in Medicine and allied specialties OPDs and admitted at Dayanand Medical College and Hospital, Ludhiana from 1/03/2021 to 28/02/2022.The patients were followed up during their hospital stay and further followed up on OPD basis for 6 weeks or till the response was achieved (whichever was early).

RESULTS: Total 123 Cases of patients presented with severe thrombocytopenia with 49 females and 74 males were including in the study with commonest age group of 19-30 years. Tropical fever was the most common cause of severe thrombocytopenia with 61 cases followed by sepsis with 23 cases. Fever was the commonest symptom followed by generalized body weakness. Bleeding manifestations were seen in 43.08% patients. Complete response was seen in 80% of the tropical fever, 43% of sepsis and 66% of ITP patients .Maximum patients with complete response were managed with steroids in ITP and symptomatically in sepsis and tropical fever.Mortality was maximum in patients with sepsis induced thrombocytopenia.

CONCLUSION: This study shows that tropical fever is the commonest diagnosis made in patients who are detected to have severe thrombocytopenia.Treatment of the patients varied with the underlying etiology.Mortality was maximum in sepsis induced thrombocytopenia.

KEYWORDS- Thrombocytopenia, tropical fever, ITP-Idiopathic thrombocytopenic purpura, sepsis, Bleeding manifestations

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INTRODUCTION

Platelets are anucleate blood cells, produced in the bone marrow by megakaryocytes. Normal platelet count is defined as 150 – 400x10^9/L. [1]

An adult produces 100 x 10^9 platelets per day which can be increased by 20-fold or more. One third of the platelets are stored in the spleen and remaining two thirds remain in the blood vessels. [2]

The main function of platelets is the maintenance of the normal homeostasis and the completion of the coagulation cascade. This process is impaired in thrombocytopenia resulting in an increased risk of bleeding events.

Thrombocytopenia, which is the deficiency of platelets, causes coagulopathy which can lead to severe bleeding manifestations resulting in significant morbidity and mortality. It is defined as platelet count less than 150x10^9/L. It can further be classified as mild – more than 70x10^9/L, moderate – 20 - 70x10^9/L and severe thrombocytopenia – less than 20x10^9/L [3].

Platelet count between 100 - 150x10^9/L can be seen in normal healthy individual [1]. When platelet counts are less than 50x10^9/L, patient can get bruised very easily and when it is less than 15x10^9/L, life threatening bleeding can be seen spontaneously. [4]

Thrombocytopenia can result from the decreased production, increased destruction, increased sequestration of platelets in spleen or combination of these factors as shown in Picture 1.

Pseudothrombocytopenia is the agglutination of platelets in the blood stream leading to false low counts in automated cell counters. It can be ruled out by peripheral film examination.

The most common cause of isolated thrombocytopenia in clinical practice is ITP (Immune Thrombocytopenia Purpura). It can be seen in any age group. In children, it usually occurs after a viral infection and resolves spontaneously within 6 months. In adults, it is often of insidious in onset and rarely resolves spontaneously. [5]

PICTURE 1: Classification of thrombocytopenia based on pathogenesis. [4]

I. Pseudo (spurious) thrombocytopenia

- Antibody induced platelet aggregation
- Platelet satellitism
- Antiphospholipid antibodies
- Glycoprotein IIb/IIIa antagonists
- Miscellaneous

II. Thrombocytopenia resulting from impaired platelet production

- Inherited platelet disorders
- Acquired marrow disorders
  1) Nutritional deficiencies and alcohol-induced thrombocytopenia
  2) Clonal hematological diseases (myelodysplastic syn-drome, leukemias, myeloma, lymphoma, paroxysmal nocturnal hemoglobinuria)
  3) Aplastic anemia
  4) Marrow metastasis by solid tumors
  5) Marrow infiltration by infectious agents (HIV, tuberculosis, brucellosis, etc.)
  6) Hemophagocytosis
  7) Immune thrombocytopenia (ITP)
  8) Drug-induced thrombocytopenia
  9) Pregnancy-related thrombocytopenia

III. Thrombocytopenia resulting from increased platelet destruction

- Immune thrombocytopenia
  1) Autoimmune thrombocytopenia (primary and secondary ITP)
  2) Alloimmune thrombocytopenia
- Thrombotic microangiopathies (TTP, hemolytic uremic syndrome [HUS])
- Disseminated intravascular coagulopathy (DIC)
- Pregnancy-related thrombocytopenia
- Hemangiomas (Kasabach-Merritt phenomenon)
- Drug-induced immune thrombocytopenia (quinidine, heparin, abciximab)
- Artificial surfaces (hemodialysis, cardiopulmonary bypass, extracorporeal membrane oxygenation)
- Type 2B von Willebrand disease

IV. Thrombocytopenia resulting from abnormal distribution of the platelets

- Hypersplenism
- Hypothermia
- Massive blood transfusions
- Excessive fluid infusions

V. Miscellaneous Causes

- Cyclic thrombocytopenia, acquired pure megakaryocytic thrombocytopenia

Immune mediated platelet destruction can occur in the presence of conditions like infections, SLE, Antiphospholipid syndrome (APLA), solid tumors or Lymphoproliferative disorders and are known as Secondary ITP. Severe thrombocytopenia occurs in 5 to 10% of patients with APLA. [6,7]

Patients with viral, bacterial, fungal and parasitic infection can have thrombocytopenia. Commonly Dengue, Scrub Typhus, Malaria, Rickettsial infections, Meningococci, Typhoid, Leptospira, Miliary Tuberculosis, HIV and certain viral infections can cause thrombocytopenia. It can be due to bone marrow suppression, increased immune destruction and due to induction of microangiopathy (infection induced Disseminated Intravascular Coagulation or Hemolytic Uremic Syndrome).[8]

Sepsis is a common cause of thrombocytopenia with increased severity and increased incidence seen in septic shock. Multiple mechanisms have been considered including platelet binding to the endothelium, leucocyte-platelet aggregate formation, immune mediated mechanisms, Hemophagocytosis , Bone Marrow suppression, Complement activation, Disseminated Intravascular Coagulation (DIC) etc. [9]

Thrombocytopenia with abnormal coagulation profile can be seen in septic DIC and in patients with liver disease due to lack of synthesis of coagulation factors and thrombopoietin. The clotting factors can be normal in up to 50% of patients of DIC and a low fibrinogen level may not always be present in DIC (less than 1 gm/L seen in less than 30% of the cases). [10]

D-ITP (drug induced thrombocytopenia) results in severe thrombocytopenia with median platelet count as $20x10^9$/L and is usually associated with mucocutaneous bleeding and constitutional symptoms like fever.[11] Drugs like Abciximab, Acetaminophen, Chlorpropamide, Cimetidine, Diclofenac, Eptifibatide, Methotrexate etc. are involved in causing thrombocytopenia. The median time from drug initiation to the development of low platelet count is 14 days, although with previous exposure, it can develop within 3 days.[12]

The onset of Heparin Induced Thrombocytopenia (HIT) is at least 5 days after starting a patient with Heparin, although if a patient had Heparin exposure in the previous 100 days, rapid onset HIT can occur on re-exposure with Heparin. The explanation for the association between recent Heparin exposure and rapid-onset HIT, is the characteristic platelet-activating HIT antibodies, which are usually no longer detectable for more than 100 days following an episode of HIT. [13]

Formation of transiently autoreactive platelet alloantibodies around 1 week following blood transfusion causes Post Transfusion Purpura (PTP). [14]

Decrease in platelet counts can be seen in post-operative patient in first 2-3 days of admission, due to combination of platelet consumption from tissue injury / wound healing, blood losses and haemodilution.

Thrombocytopenia can develop in conjunction with a large venous or arterial thrombosis due to HIT, APLA syndrome and massive clot thrombocytopenia, where pulmonary embolism can have large numbers of platelets incorporated into an extensive thrombi. [15]

Hematologic disorders like Megaloblastic anaemia are one of the causes of thrombocytopenia due to impaired DNA synthesis resulting in ineffective Erythropoiesis.

Haematological malignancies can be a cause of thrombocytopenia and may present acutely with severe infections or anaemia. Myelodysplastic syndromes can also present with isolated thrombocytopenia as the presenting symptom needing intensive care admission.

Thrombocytopenia is often characterized by bleeding from small vessels which can present as petechiae over the skin, haemorrhage from mucosa of gastrointestinal and genitourinary tract. Intracranial haemorrhage is a serious consequence in thrombocytopenic patients.[16]

Thrombocytopenia associated with fever helps to narrow down the differential diagnosis. Patient presents with varied clinical manifestations which can range from asymptomatic or simple viral

illness to severe circulatory shock. Timely identification and treatment of underlying cause can prevent bleeding manifestations, reduce the requirement of steroids and platelet transfusions and can impact the mortality of the patients.

In case of drug induced thrombocytopenia, offending drug needs to be stopped. In patients with liver disease, coagulation profile needs to be corrected while in sepsis, patient primarily requires the management of infections in order to correct the thrombocytopenia. If associated with any hematological malignancy, patient is to be managed on the lines of underlying malignancy. In cases of tropical fever, gradual rise of platelets is usually seen with symptomatic management.

The most commonly accepted platelet count threshold for transfusions is a level of $10x10^9$/L without any risk factors and 20×10^9/L in patients with risk factors for bleeding. These risk factors include sepsis, DIC, severe hepatic or renal dysfunction. [17]

Treatment of ITP Is initiated when platelet count falls below 20-50x 0^9/L. Corticosteroids remain the mainstay of treatment for both primary ITP and Lupus associated ITP. Splenectomy, Disease Modifying anti-Rheumatic drugs like Azathioprine, Mycophenolate mofetil, Calcineurin inhibitors and Dapsone are considered as a second line treatment option for primary ITP. Rituximab has been very commonly studied as a steroid-sparing therapy for the treatment of primary and secondary ITP. [18] Immunosuppressive therapy, either alone or in combination with glucocorticoids, can be given for nephritis and neurological symptoms. [19,20] IVIG is to be reserved for patients with severe bleeding. [21,22] Rituximab (B-cell targeted therapy) is effective for patients with refractory SLE (especially those with nephritis and severe thrombocytopenia). [23]

Thrombopoietin Receptor Agonists (TPO-RA) are another line of drugs that can be used in Thrombocytopenia. Romiplostim, a “peptibody”, contains four copies of a 14-amino-acid peptide grafted onto an Ig-Fc domain. It binds to a region of the Thrombopoietin receptor leading to increase in platelet counts. This drug has been approved by FDA in 2008 in patients with ITP in whom other modalities of treatment have

failed.[24] Eltromobopag, was also approved in 2008 by FDA for the similar indications. [25] It binds to the transmembrane domain of the TPO receptor thus increasing the platelet count.

In APLA syndrome, immunosuppressive treatment increases the platelet count. Glucocorticoids are effective in 15% of patients. [26] IVIG and immunosuppressive drugs such as Azathioprine and Cyclophosphamide can be used in patients with severe bleeding. Splenectomy should only be preferred in patients with severe bleeding. Rituximab has been used to treat refractory thrombocytopenia in patients with APS (Antiphospholipid Syndrome).[27,28] TPO receptor agonists may increase risk of thrombosis in patients with APS and SLE. These diagnoses in association with ITP were accepted as exclusion criteria in some randomized controlled studies of TPO-receptor agonists. [29]

IVIG blocks the macrophage Fc receptors thus decreasing the destruction of platelets. It has become first line therapy in children and can also be used in adults.[30]

American Society of Hematology guidelines 2011 recommends IVIG as a first line therapy in patients with ITP secondary to HCV, because glucocorticoids can increase the viral loads.[31] Thrombopoietin Receptor Agonists (TPO-RA) can cause abdominal thrombosis in HCV patients.[32]

Thrombocytopenia secondary to tuberculosis improves with anti-tubercular treatment.

Thrombocytopenia has been demonstrated as a poor prognostic factor in critically ill patients. Severe thrombocytopenia can be suggestive of severe underlying disease and can be an indicator of mortality.

The present study was undertaken to assess the etiology, clinical and laboratory profile, response to treatment and outcome of severe thrombocytopenia.

AIMS AND OBJECTIVES OF THE STUDY

- To study the clinical profile of patients presenting with severe thrombocytopenia
- To study the outcome of patients presenting with severe thrombocytopenia.

REVIEW OF LITERATURE

Thrombocytopenia is a major concern in intensive care medicine. Its prevalence varies between 13% and 58% depending on the clinical setting and definition. [33]

Tropical fever is one of the predominant etiologies of severe thrombocytopenia. India being a tropical country, has high incidence of infections like Malaria, Leptospirosis, Dengue fever, Scrub fever, Typhoid fever, Chikungunya which can cause life threatening organ dysfunction and other complications. [34]

A study of 100 cases of thrombocytopenia done by Sridhar et al [35] found that Dengue was the most common cause followed by sepsis. The mean age of the patients in the study was 42.92 years with Males being more than female. Bleeding manifestations were seen in one fifth (23%) of the cases. Steroids were given in 11% of the patients. Mortality was highest in patients with sepsis induced thrombocytopenia.

A clinical study of febrile thrombocytopenia was done by Lakshmikumar et al [16], in which 190 cases of fever with thrombocytopenia were taken. Mean age was 41.11 years with 60.5% males and 39.5% females. Infection was the commonest cause of thrombocytopenia (68.9%) followed by septicaemia (9.5%) and the most common infection was Dengue (68.9%).

PICTURE 2: Distribution of patients according to etiology in Lakshmikumar et al [16] study**.**

Diagnosis	No of patients
Dengue fever	131(68.9%)
Septicaemia	18(9.5%)
Viral fever other than dengue	17(9.0%)
Malaria	16(8.4%)
HIV	4(2.1%)
Typhoid fever	4(2.1%)

Among 190 cases, all of the them presented with fever, 147 complained of headache, 140 of vomiting, 139 of arthralgia, 137 of retro-orbital pain, 125 of myalgias, 100 of rashes, 94 of pain abdomen and 83 of bleeding manifestation.

PICTURE 3: Distribution of patients according to bleeding manifestation in Lakshmikumar et al [16] study**.**

Bleeding manifestation	No. of patients
Venepuncture bleed	32(16.8%)
Gum bleed	28(14.7%)
Hematuria	21(11.1%)
Petechiae	17(8.9%)
Epistaxis	15(7.9%)
Haematemesis	3(1.6%)
Malena	3(1.6%)

In 131 cases of Dengue fever, 28 had gum bleed, 21 had hematuria, 17 had venepuncture, 16 had petechiae, 5 had epistaxis, 3 had haematemesis and 3 had malena. Total bilirubin was elevated in 33.7% cases. SGOT and SGPT were elevated in 39.5% and 37.4% cases respectively. Urea was elevated in 20.5% cases and creatinine in 23.7% cases. Out of the 190 cases, 179 (94.2%) were discharged, 11 (5.8%) expired during hospital stay.

Etiological spectrum of thrombocytopenia was studied by Gamit et al [36]. It was observed to be present more in males (55.75%) than females (44.25%). Most patients belonged to the age group of 18-29 years (45%) and the most common etiology was found to be Dengue fever in 167(41.75%) cases, followed by Malaria in 106 (26.50%) cases, chronic liver disease in 58 (14.5%) cases, septicemia in 29 (7.25%) cases, aplastic anaemia in 8 (2%) cases, DIC in 17 (4.25%) cases and other causes in 15 (3.75%) cases.

A study was done by Magdalene et al [37] on 200 cases of thrombocytopenia during 2 years period. Most common cause of thrombocytopenia was found to be Immune Thrombocytopenic Purpura

in 55 cases (27.5%) followed by infections in 42 cases (21%). Hematological malignancy were seen in 21 cases (10.5%), Megaloblastic anaemia in 19 (9.5%) and drug induced thrombocytopenia was seen in 12 cases (6%). Other causes include aplastic anaemia, hypersplenism, alcoholism, immunological disorder, Paroxysmal Nocturnal Hemoglobinuria, valvular heart disease, pregnancy, iron deficiency anaemia, Gaucher's disease, DIC due to snake bite, artificial valve prosthesis and massive blood transfusion.

Clinical profile of thrombocytopenia was studied by Mural et al [38]. A total 37 patients were studied. The highest incidence of thrombocytopenia belonged to the age group 18-30 years (36%) and was more in women (64%) as compared to men (36%). The most common cause of thrombocytopenia in this study was septicemia (17%) followed by Megaloblastic anaemia (14%) and Dengue fever (14%). Almost 8% patients were reported with Malaria. 19% of the patients showed bleeding manifestations. Common etiologies with severe thrombocytopenia (less than $20x10^9$/L) were Dengue (45%), Megaloblastic anaemia (27%), Enteric fever (18%) and ITP (9%). Most common bleeding manifestations was seen in skin and mucous membrane (43%) followed by gum bleeding (29%), malena (14%) and intracranial bleeding (14%). 20% patients had pancytopenia, 28% patients had leucopenia with thrombocytopenia, 39% patients had anaemia with thrombocytopenia and 13% patients had selective thrombocytopenia. Out of 7 patients of pancytopenia, 4 patients had megaloblastic anaemia, 2 had retroviral disease and 1 patient had disseminated tuberculosis.

Another study done by Vimal et al [39] on clinicopathological spectrum of thrombocytopenia on 120 patients with 71 males (59.2%) and 49 females (40.8%). Severe thrombocytopenia (less than 50,000/mm^3) was seen in 20% patients. The etiological diagnosis of patients included in the study were Dengue (21.6%), Malaria (6.7%), Enteric fever (5.9%), septicemia (5%), CLD (16.7%), CKD (3.4%), Diabetes (7.7%), malignancy (1.67%) , CAD (3.4%), pregnancy (5%), haematological disorder (18.4%) and miscellaneous (5%). 54 patients

(45%) presented with fever and 16 (13.3%) with bleeding manifestations.

Zhang Man-Ka et al [40] studied 737 adult patients in intensive care unit and found that infections (32.7%) were the most frequent cause associated with thrombocytopenia, followed by septic shock (3.93%) and blood loss (2.99%).

Maheshkumar et al [41] studied 920 cases of thrombocytopenia, most common cause was found to be infections (42.61%) followed by anaemia (30.33%). ITP was seen in 0.33% only. Among infection, most common cause is Dengue (18.80%) followed by Malaria (10.76%) and septicemia (4.57%). Most common age group was 35-54 years. There were 546 (59.35%) males and 374 (40.65%) females in the study. Severe thrombocytopenia was seen in 47 patients, out of whom, 15 patients had bleeding episodes.

Yadav et al [42] found septicaemia (24.1%) as the most common cause of thrombocytopenia followed by Megaloblastic anaemia (13.10%). Other causes include alcoholic liver disease, non-alcoholic liver disease and malignancies. 16.27% patients presented with bleeding manifestation. Bleeding events seen more in severe thrombocytopenia (27.27%) (p-0.008). Petechiae was seen in 66% cases with severe thrombocytopenia (p=0.025). The overall mortality was seen in 19.84% cases and in 29.54%, 22.22% and 14.96% cases respectively among the severe, moderate and mild groups. There were 135 (53.57%) males and 117 (46.43%) females. The most common cause of death was septicaemia (40%).

Boxer et al [43] studied the various etiologies of thrombocytopenia and found that 35 patients had a primary hematologic cause. Most common hematologic diagnosis was ITP (n=12), HIT (n=6), in vitro platelet clumping (n=4), Acute Progranulocytic Leukaemia (n=4), and Thrombotic Thrombocytopenia Purpura (n=3). 51 patients had secondary causes for thrombocytopenia. Most common non hematologic etiologies were liver disease (n=21), bacterial sepsis (n=12), and oral medication (n=7).

In a study by Fountain et al [44], patients with moderate thrombocytopenia (platelet count less than $100x10^9$/L or a >50% relative decline) were compared to patients with mild thrombocytopenia ($100\text{-}150x10^9$/L or <50% relative decline). Severe sepsis and antibiotic use were associated with moderate thrombocytopenia. As reported by them, most commonly used medications causing thrombocytopenia include Heparin, antibiotics, Proton Pump Inhibitors and Antiplatelet therapy. Sepsis was caused by respiratory (13%), genitourinary (24%), gastrointestinal (5%) and skin and soft tissue (7.7%) infections. Haematological complications and outcome metrics including transfer to intensive care unit, death during admission and length of stay were all associated with moderate thrombocytopenia.

Clinical presentation of thrombocytopenia varies from being asymptomatic to patient presenting with bleeding episodes.

Makroo et al [45] suggested that bleeding occurs more often in patients with severe thrombocytopenia. High-risk patients having platelet count <20,000/mm^3 and risk of bleeding requires urgent platelet transfusion. Patients with platelet count 21-40,000/mm^3 are in a moderate risk category and require platelet transfusion only if they have any haemorrhagic manifestations and other superadded conditions.

Piel-julian et al [46] did a cross sectional study and determined that platelet counts < $20x10^9$/L and < $10x10^9$/L were threshold with major increased risks of any bleeding events. Platelet count, female sex, exposure to NSAIDs and anticoagulant use should be considered to assess the risk of any bleeding.

There are 5 studies which reported the correlation between low platelet counts and bleeding. Khellaf et al [47] reported a moderate correlation between a platelet count <$20x10^9$/L and bleeding ($r = -0.40$; $p<0.01$).

Page et al [48] did not observe any correlation between thrombocytopenia (<30 $x10^9$/L) and bleeding.

Pansy et al [49] observed an association between platelet count at diagnosis and bleeding score among treated patients ($p=0.022$), and at days 5-8 among untreated patients ($p = 0.015$); however, there was not a consistent correlation across all study visits or patient groups.

In a large follow up study of 292 patients of Thrombocytopenia treated with Romiplostim by Kuter et al [50], 51% of all bleeding events (mostly mild) occurred at platelet count levels below 50Sx10^9/L. In that study, 61% of severe bleeding events occurred at platelet counts <20x10^9/L and none were considered life-threatening. Overall, most bleeding events occurred with platelets <30x10^9/L with frequent exceptions, including one patient with ICH and a platelet count of 120x10^9/L as observed by Shirasugi et al [51].

Latvala et al [52] concluded in their study that abnormal platelet and leukocyte levels were common, especially in the anaemic alcoholics. Reticulocytosis (37%), thrombocytopenia (41%), and combined cytopenias (34-38%) were also common findings in the alcoholic patients.

Early identification and intervention of underlying condition and transfusion of platelets are required to prevent fatal outcome.

Mak et al [53] performed a retrospective analysis of bone marrow examination in the management of Chinese adult patients less than 60 years of age with isolated thrombocytopenia at presentation. Total 83 patients were studied over a period of almost 4 years. All 83 patients had bone marrow findings of active marrow suggesting causes related to peripheral destruction. Eighty one of the 83 patients, after a follow up of 20 months were diagnosed as having ITP. The remaining two patients were diagnosed with Systemic Lupus Erythematosus. All the patients had normal bone marrow with normal or increased megakaryocytes and without a single case of Myelodysplasia, Aplastic anaemia, or infiltration with Lymphoma or solid neoplasms.

Westerman et al [54] have also reported 66 patients who had undergone bone marrow examination for the investigation of isolated thrombocytopenia at presentation. Among them, 61 showed normal or increased numbers of megakaryocytes with other hematopoietic lineages being normal. In the remaining patients, subsequent follow up suggests that all of them had chronic ITP despite the presence of marrow hypocellularity or mild granulocyte dysplasia at presentation. They concluded that bone marrow examination for diagnosis of isolated

thrombocytopenia is not useful provided that a thorough clinical history and physical examination are undertaken.

Management of thrombocytopenia is largely directed towards treating the underlying cause.

Thrombocytopenia and bleeding are the most common complication observed in Dengue patients. According to the guidelines, platelet transfusion is indicated in case of active bleeding or severe thrombocytopenia. [55]

There are numerous risks associated with prophylactic platelet transfusion including allo-immunisation, platelet refractoriness, allergic reactions, febrile non haemolytic reactions, bacterial sepsis and less commonly Transfusion Related Acute Lung Injury (TRALI).

Slichter et al [56] suggested that patients with platelet counts of $20x10^9$/L can be given platelet transfusions prophylactically. In this study of 92 patients, there were 34 cases of Acute Myeloid Leukemia and 57 cases of Acute Lymphocytic Leukemia. In patients with platelet count $1x10^9$/L, gross bleeding was seen in 33% of the days. In patients with platelet counts between $5x10^9$/L and $20x10^9$/L, gross bleeding occurred on only 3% of the days.

Most of the evidence for platelet transfusion therapy derives from studies done in patients with Chemotherapy induced thrombocytopenia.

Three randomized trials enrolling 492 patients showed no difference in bleeding with a prophylactic platelet transfusion trigger of $10x10^9$/L or $20x10^9$/L. Based on this data, a platelet trigger of $10x10^9$/L has been generally adopted for prophylactic platelet transfusion in stable haematology/oncology patients. [57,58,59]

Evidence to guide platelet transfusion in non-oncology patient is lacking.

A retrospective study was done by Arnold et al [60], examining 76 platelet transfusions administered to 27 patients. The threshold platelet count was $33x10^9$/L for prophylactic transfusions, $46x10^9$/L for patients planned for invasive procedures and $51x10^9$/L for the treatment of bleeding. The platelet count increased by a median of $14x10^9$/L after

one platelet transfusion. According to the published guidelines, a platelet count trigger of $50x10^9$/L for patients with DIC or massive transfusion and 50 - $100x10^9$/L for patients in whom an invasive procedure is planned.

Peter-Salonen et al [61] studied a corrected thrombocyte increment for recovery in patients receiving platelet transfusions. A total of 31 patients with 108 transfusions were evaluated in this study. The mean recovery at the one hour post transfusion time point was $8.6x10^9$/L with fresh platelets and $5.9x10^9$/L with stored platelets. Better recovery was seen significantly with freshly prepared platelets. The transfusion with fresh platelet in patients with signs indicating enhanced platelet destruction like fever, splenomegaly and DIC, gave a significantly better recovery as compared to stored platelet concentrates ($p=0.028$). In absence of such signs the recovery produced by fresh platelet concentrates was not significantly higher than with stored concentrate.

There are 2 randomized prospective transfusion trials comparing therapeutic and prophylactic transfusions of platelets in thrombocytopenic patients.

In one study done by Murphy et al [62], 56 children with Acute Leukemia were randomly assigned to receive platelets prophylactically at a platelet count of $20x10^9$/L irrespective of the clinical events and therapeutically only when significant bleeding had occurred (nasal or oral bleeding requiring packing, gross gastrointestinal bleeding, gross genitourinary bleeding, central nervous system bleeding or any bleeding requiring red cell transfusion). There were significantly fewer bleeds per 100 patient months in the prophylactic group but the total days of bleeding per 100 patient months were same in both the groups. The prophylactic group received almost 3 times the transfusion received in therapeutic group. Survival rates were found to be same in both the groups.

The second study done by Solomon et al [63] evaluated the use of platelets in 29 adults during intensive induction chemotherapy for Acute non-Lymphoblastic Leukemia. The prophylactic group received transfusions when platelet counts were below $20x10^9$/L. On the other

hand, therapeutic group received transfusions when they had significant bleeding or had a platelet count below $20x10^9$/L preceeded by a 50% drop in the count in the prior 24 hours. It is, thus, conceivable that some patients in the therapeutic group were not bleeding, and yet they received platelets. Similar to the other study, platelet use was significantly higher (approximately twice as much) in the prophylactic group compared with the therapeutic group, whereas other study parameters were the same between the two groups.

Chang et al [64] studied the response of steroids in 257 patients with ITP. Response to steroids was seen in 184 patients. Complete response was seen in 120 patients (65.2%), partial response in 43 patients (23.3%) while 21 patients (11.4%) were refractory to steroids. Among patients with complete response, 76 (63%) patients relapsed in a median of 9.5 months. After relapse or steroid failure, 57(49%) received azathioprine treatment and 38 (32%) underwent splenectomy. Response rate was 71.4% for azathioprine and 91.4% for splenectomy. Both azathioprine and splenectomy are effective treatment after steroid failure. Rituximab was effective in 8 of 10 patients and was suggested as a reasonable second-line treatment option.

Mazza et al [65] studied 124 patients of ITP retrospectively in their multicentre study. 55 patients were given Romiplostim and 69 were given Eltromobopag. Mean age, number of young patients (<60 years), time from primary diagnosis of ITP to TPO-RA treatment, and previous lines of therapy were similar in both groups. The mean duration of response was found to be 30 months in patients taking Romiplostim and 15 months in patients with Eltromobopag.

Serebruany et al [66] studied the preclinical testing on healthy volunteers and found that Eltromobopag has high drug bioavailability and efficacy in raising platelet counts. The 10-day treatment with Eltromobopag increased platelet count up to 1.5 times. The drug is usually well tolerated and serious side effects and discontinuation are rare. However its long term safety is not known. The increased risk of thrombosis and thus cardiovascular events and stroke, can be the major obstacle for the success of Eltromobopag.

The presence of thrombocytopenia can be an omnious sign upon outcome analysis

Crowther et al [67] studied the impact of thrombocytopenia in critically ill patients in medical-surgical ICU. 261 patients were included in the study, out of which 121 patients had thrombocytopenia on admission and 59 acquired during hospital stay. Patients who developed a platelet count less than $150x10^9$/L during their ICU stay had higher ICU and hospital mortality (p=0.03 and 0.005 respectively), required longer mechanical ventilation (p=0.005) and red blood cell transfusions (p=0.004) than patients who did not develop thrombocytopenia.

Strauss et al [68] did a prospective observational study in the patients admitted with normal platelet count and intensive care unit stay of >48 hours, for 13 months. The prevalence of intensive care unit acquired thrombocytopenia was 44% (n=64). The mortality was found to be more in the thrombocytopenic patients (31%) as compared to non-thrombocytopenic patients (16%). Patients with platelet $<100x10^9$/L (p<0.01) and those with a 30% or more drop in platelet count are found to have higher mortality.

Koebnick et al [69] found that Intensive care unit mortality was 31% in thrombocytopenic patients and 16% in non-thrombocytopenic patients (p=0.03). Mortality was higher in patients with platelet count less than $10x10^9$/L (p < 0.01) and in patients with a drop in platelet count of > 30% (p < 0.01). A logistic regression analysis identified septic shock, a higher Acute Physiology and Chronic Health Evaluation II score at admission and a drop in platelet count exceeding 30% as independent risk factors for intensive care unit death.

Stephan et al [70] did a prospective study in 147 surgical ICU patients admitted during a 6 month period. Thrombocytopenia occurred in 52 patients (35%) with a mortality rate of 38% compared to 20% mortality rate in non-thrombocytopenic patients (p=0.02). Sepsis, episodes of bleeding or transfusions and an acute physiology and chronic health evaluation (APACHE II) score of > 15 were the independent risk factors identified for thrombocytopenia. DIC was found in 40% of

thrombocytopenic patients, elevated platelet associated IgG in 33% and Hemophagocytic histiocytes in 67%.

Nijsten et al [71] performed a retrospective study in patients admitted to the ICU for at least 4 days during a 7 year period and concluded that a blunted or absent rise in platelet count in critically ill patients is associated with increased mortality. In patients who recover uneventfully from an inflammatory insult, the platelet displays a bimodal response with an initial decrease below baseline values for the first days, followed by an increase above the normal range after 1 week.

MATERIALS AND METHODS

A prospective study was conducted in patients presenting with severe thrombocytopenia in Medicine and allied specialities OPDs and admitted at Dayanand Medical College and hospital, Ludhiana From 1/03/2021 to 28/02/2022. The patients were followed up prospectively during their hospital stay and further followed up on OPD basis for 6 weeks or till the response was achieved (whichever was early). The demographic data, including patient age, sex, and diagnosis at the time of admission, clinical and laboratory findings was collected and recorded in a proforma for analysis and interpretation of data.

Data and observations describing possible complications of thrombocytopenia, such as bleeding manifestations (Multiple bleeding events at the same location was counted only once for each patient), shock was collected and recorded in the proforma.

The data of various types of intervention including number and types of transfusions (random donor platelets/single donor platelets, fresh frozen plasma, and packed red blood cells), drugs like steroids, Thrombopoietin Receptor Agonists, IVIG, Anti-D given to the patient was also recorded. The response of intervention was measured in terms of complete response, partial response and no response. Response was defined as platelet count more than or equal to $30X10^9$/L and at least 2 fold increase in the baseline value and absence of bleeding. Complete response was defined as platelet count more than or equal to $100x10^9$/L and absence of bleeding and no response was defined as platelet count less than $30x10^9$/L or less than 2-fold increase of baseline platelet count or bleeding. [72]

The outcome of the patients in terms of duration of hospital stay, need for ICU care, need for mechanical ventilation was observed and recorded.

SOURCE OF DATA

Patients with severe thrombocytopenia attending Medicine and Allied specialties OPDs, admitted in Medicine and Allied specialties wards at

Dayanand Medical College and hospital, Ludhiana from 1/03/2021 to 28/02/2022.

METHOD OF COLLECTION OF DATA

Cases were selected on the basis criteria set in inclusion and exclusion criteria for the study

Inclusion Criterion:

1) Patient with severe thrombocytopenia (less than $20x10^9$/L)

2) Patient age more than or equal to 18 years

Exclusion Criterion:

1) Patients with mild and moderate thrombocytopenia

2) Patients not willing to take part in the study

3) Age less than 18 years.

4) Patients diagnosed with any malignancy

OBSERVATIONS & RESULTS

The study was conducted at Dayanand Medical College and Hospital, Ludhiana in the Department of Medicine. This study was conducted prospectively from March 2021 to February 2022 with follow up of 6 weeks.

Out of a total of 123 patients, 7 patients were lost were lost to follow up of the response to treatment, so the response to treatment was recorded in 116 patients.

Table 1: Age wise distribution of the patients presenting to the hospital with severe thrombocytopenia during the years 2021-2022

Age groups (in years)	No. of cases	Percentage
19-30	30	24.4%
31-40	27	22.0%
41-50	23	18.7%
51-60	16	13.0%
61-70	17	13.8%
>70	10	8.1%
Total	123	100.0%

Maximum patients presented with severe thrombocytopenia belonged to the age group of 19 – 30 years (24.4%) followed by 31 – 40 years (22%).

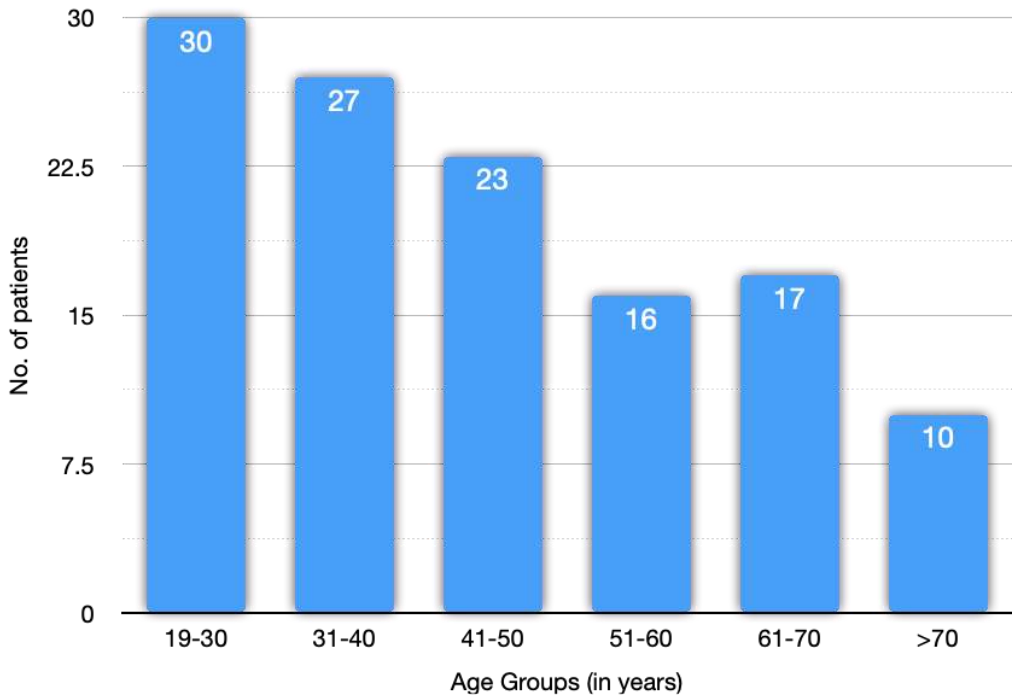

Figure 1: Age wise distribution of the patients presenting to the hospital with severe thrombocytopenia

Table 2: Distribution of patients presenting to the hospital with severe thrombocytopenia during the years 2021-2022 on the basis of gender

Sex	No. of cases	Percentage
F	49	39.8%
M	74	60.2%
Total	123	100.0%

Out of the enrolled 123 patients, majority were males (60.2%) as compared to females (39.8%).

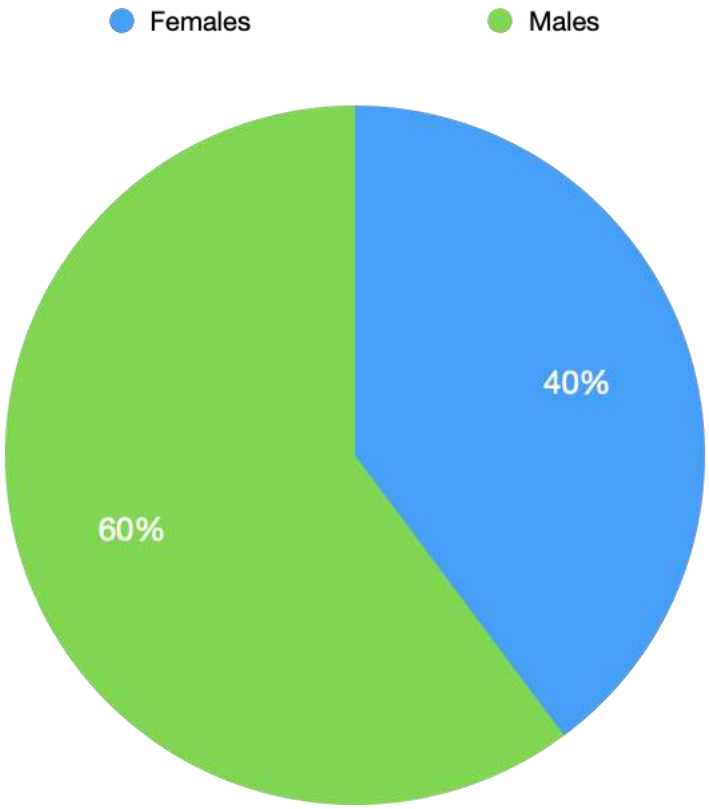

Figure 2: Bar graph showing distribution of patients presenting to the hospital with severe thrombocytopenia during the years 2021-2022 on the basis of gender

Table 3: Distribution of etiology of patients presenting to the hospital with severe thrombocytopenia during the years 2021-2022

Etiology	No. of cases	Percentage
Tropical fever	61	49.59%
Sepsis	23	18.7%
Autoimmune	3	2.44%
Drug Induced	1	0.81%
ITP	18	14.63%
Tuberculosis	3	2.44%
Megaloblastic anaemia	2	1.63%
Multifactorial	12	9.76%

Half of the patients presented with severe thrombocytopenia were diagnosed with tropical fever (49.59%) followed by sepsis (18.7%). ITP was found to be the 3rd most common cause (14.63%) of severe thrombocytopenia. Other causes found in this study included autoimmune (2.44%), tuberculosis (2.44%), drug induced (0.81%) and megaloblastic Anaemia (1.63%). 12 patients (9.76%) were found to have multiple etiologies causing severe thrombocytopenia.

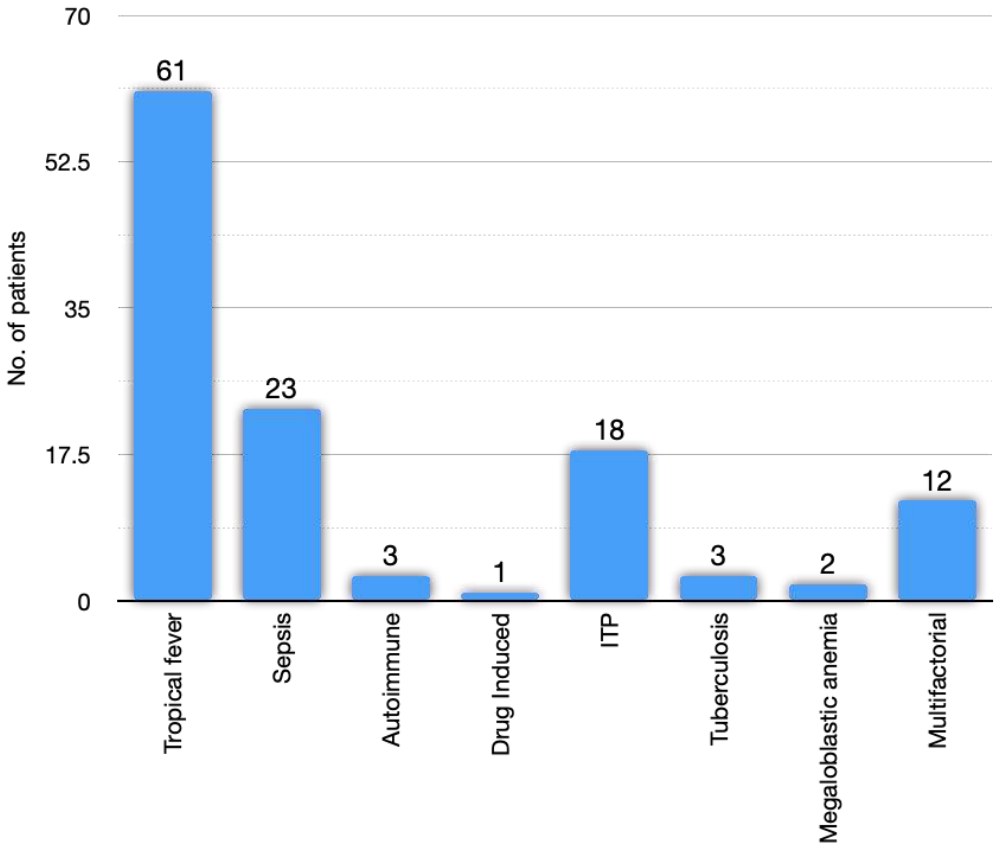

Figure 3: Bar graph showing distribution of etiology of patients presenting to the hospital with severe thrombocytopenia during the years 2021-2022

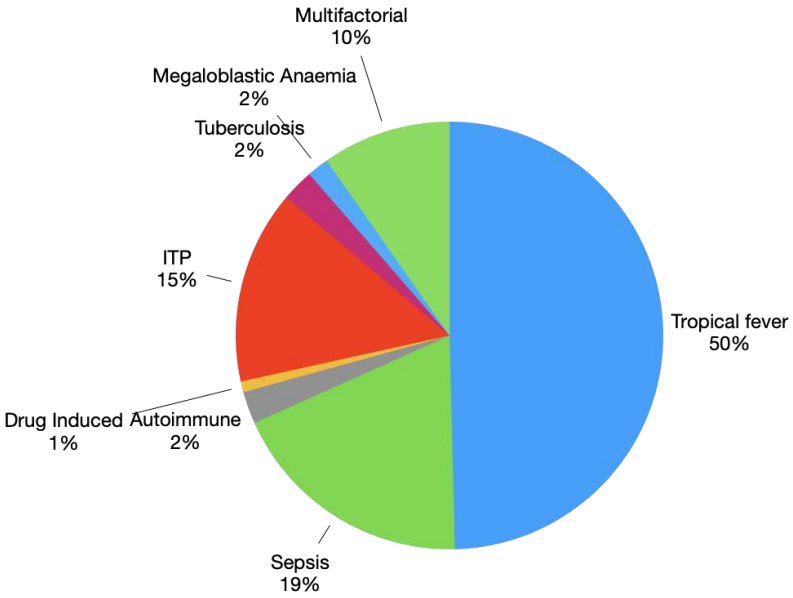

Figure 4: Pie chart showing distribution of etiology of patients presenting to the hospital with severe thrombocytopenia

Table 4: Distribution of patients with severe thrombocytopenia diagnosed with multiple etiologies

Etiology	No. of cases	Percentage
Autoimmune + sepsis	1	8.33%
ITP + Tropical fever	4	33.33%
ITP + Autoimmune	1	8.33%
ITP + Tropical fever + Autoimmune	1	8.33%
Tropical fever + sepsis	1	8.33%
Tropical fever + autoimmune+ HLH	1	8.33%
Sepsis + HLH	2	16.67%
Tropical fever + sepsis + HLH	1	8.33%
Total	12	100%

The table shows the distribution of the 12 patients in whom multiple etiologies were found to be responsible for causing severe thrombocytopenia. Tropical fever has been seen together with ITP, autoimmune etiology as well as with sepsis. There was a patient diagnosed with autoimmune etiology along with a soft tissue sepsis. Hemophagocytosis has been seen in four patients.

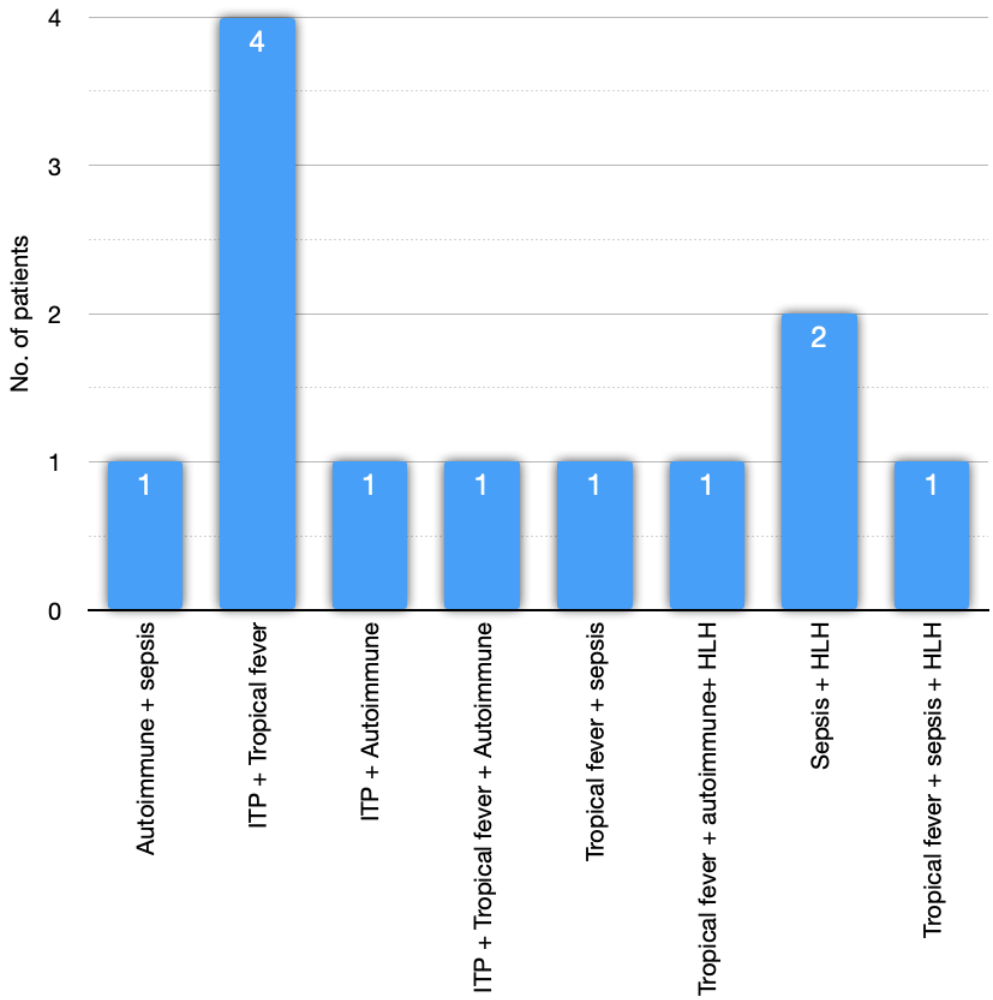

Figure 5: Bar graph showing distribution of patients with severe thrombocytopenia diagnosed with multiple etiologies

Table 5: Distribution of patients with severe thrombocytopenia according to sub-etiology of tropical fever

Sub-etiology	Total no. of cases	Percentage
Dengue	45	73.77%
Leptospira	3	4.92%
Scrub	1	1.64%
Malaria	0	0.00%
Enteric fever	0	0.00%
Hepatitis - A	1	1.64%
Dual tropical infections	5	8.20%
Tropical fever with other etiologies	6	9.84%
Total	61	100%

Total 61 patients were having thrombocytopenia due to tropical fever. Dengue was found to be the most common cause among the tropical fever (73.77%). Dual infections were found to be the next most common cause (8.20%) of the tropical fever. Tropical fever in association with other etiologies was found in 6 patients (9.84%)

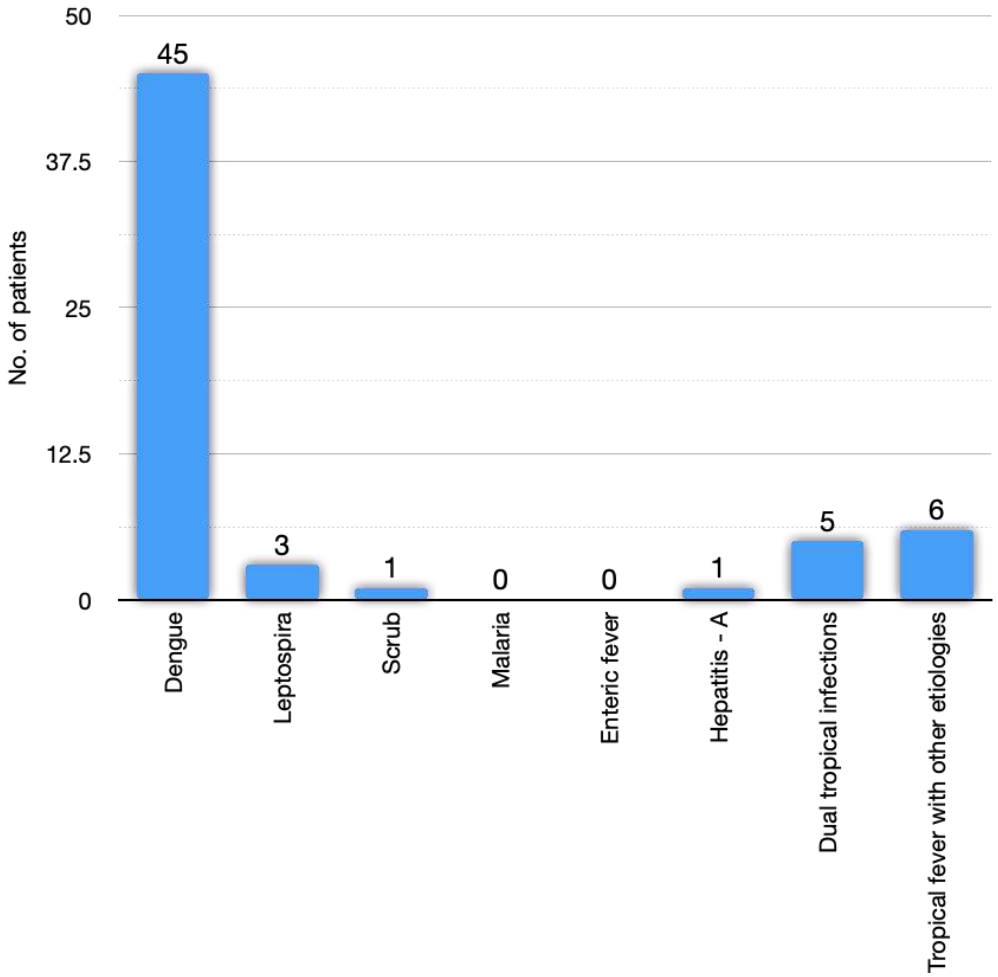

Figure 6: Bar graph showing distribution of patients with severe thrombocytopenia according to sub-etiology of tropical fever.

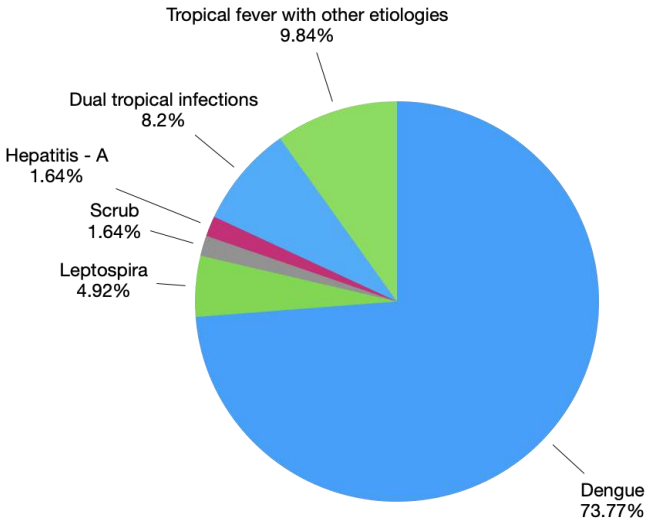

Figure 7: Pie chart showing distribution of patients with severe thrombocytopenia according to sub-etiology of tropical fever.

Table 6: Distribution of patients having dual Tropical fever infections.

`	Total no. of cases	Percentage
Dengue + Malaria	1	20%
Dengue + Scrub	2	40%
Dengue + Leptospira	1	20%
Leptospira + Enteric	1	20%
Total	5	100%

Out of a total of 5 patients with dual Tropical fever infections, most common combination was of Dengue fever with Scrub Typhus (40%).

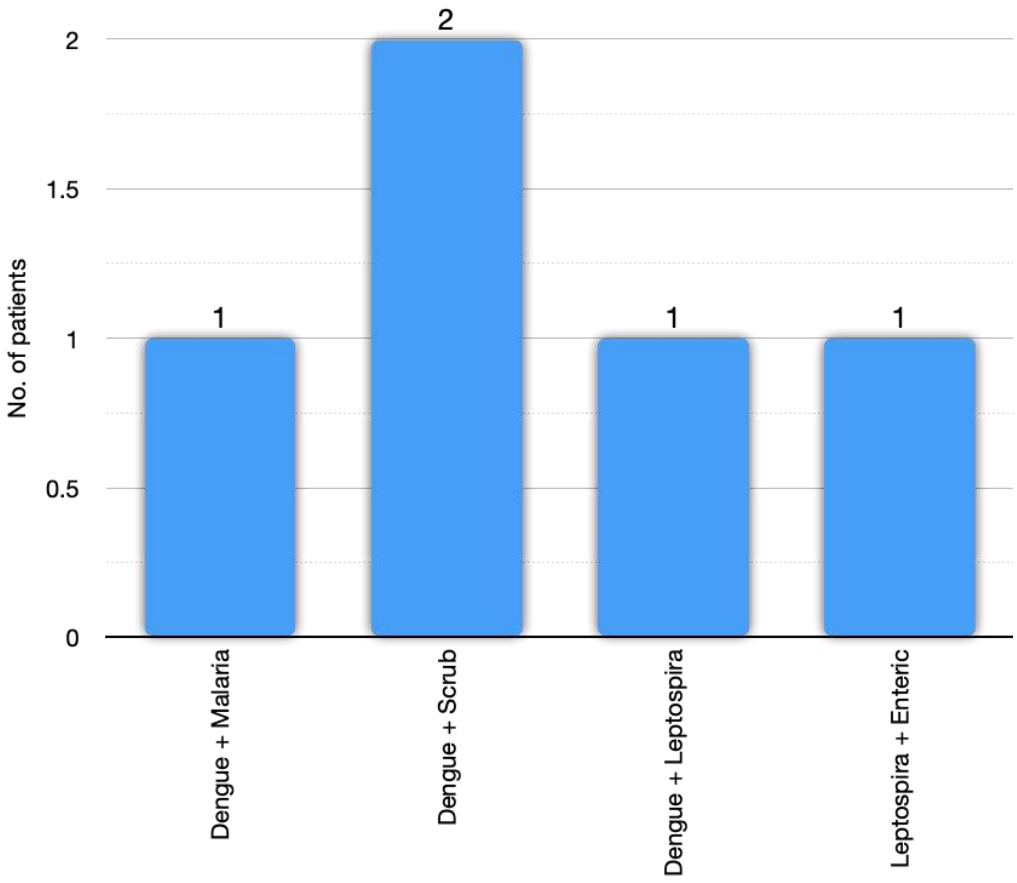

Figure 8: Bar Graph showing distribution of patients having dual tropical fever infections.

Table 7: Distribution of patients presenting with severe thrombocytopenia with different source of sepsis

	No. of Patients	Percentage
Gastrointestinal	8	34.78%
Genitourinary	9	39.13%
Infective Endocarditis	1	4.35%
Respiratory	1	4.35%
Respiratory, Genitourinary	2	8.70%
Not Known	2	8.70%

Genitourinary infections (39.12%) were the most common source of Sepsis in our patients. Approximately one third of the patients (34.78%) had Sepsis due to gastrointestinal infections.

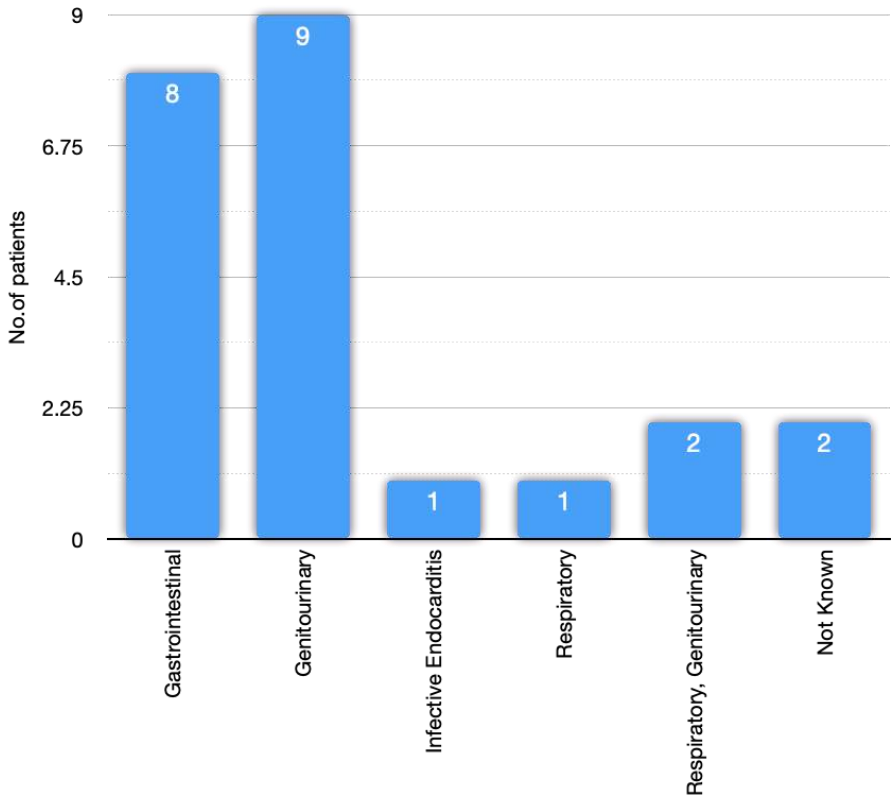

Figure 9: Bar Graph showing distribution of patients with severe thrombocytopenia with different source of sepsis

Table 8: Distribution of patients with severe Thrombocytopenia according to history of Alcohol

Alcohol history	Total no. of cases	Percentage
Yes	12	9.8 %
No	111	90.2 %
Total	123	100 %

Out of the total 123 patients, 9.8 % patients were found to have alcohol history while 90.2% has no alcohol history.

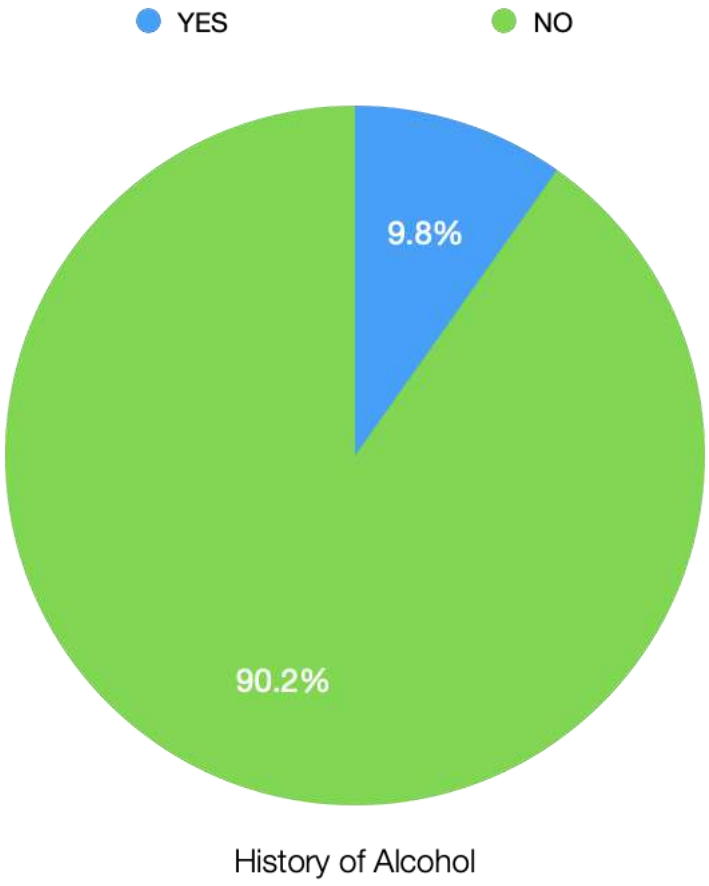

Figure 10: Pie chart showing distribution of patients with severe Thrombocytopenia according to history of Alcohol

Table 9: Distribution of patients with positive history of Alcohol according to Etiology of Severe Thrombocytopenia

Etiology	No. of patients	Percentage
Sepsis	9	75%
Tropical fever	2	16.67%
ITP	1	8.33%

Among the patients with positive Alcohol history, three fourths of the patients had Sepsis induced Thrombocytopenia.

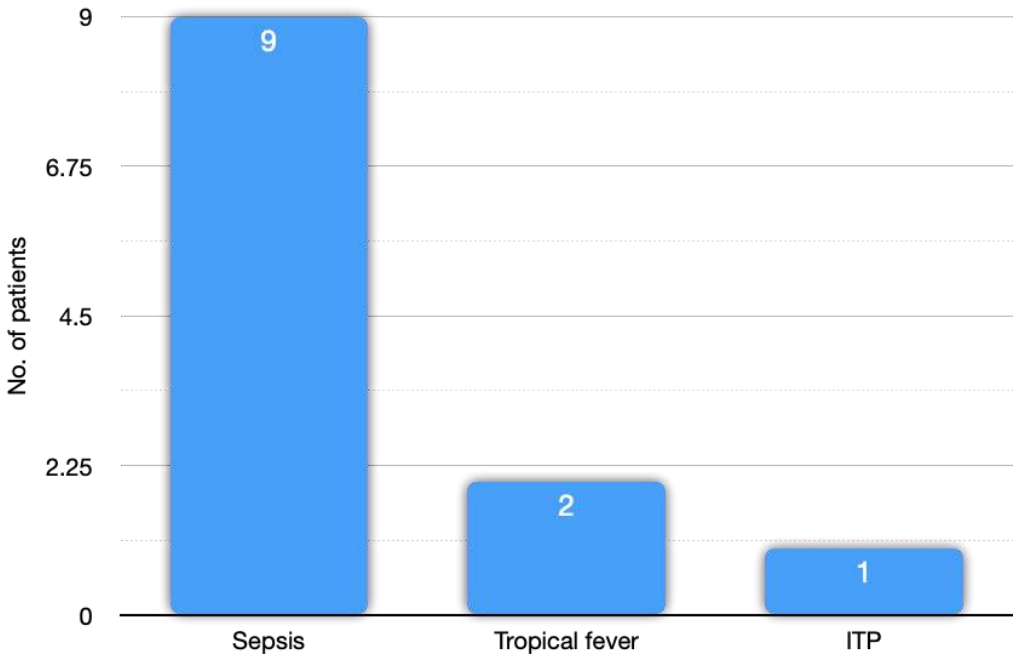

Figure 11: Bar graph showing distribution of patients with positive history of Alcohol according to Etiology of Severe Thrombocytopenia

Table 10: Distribution of comorbidities associated with patients presented with severe thrombocytopenia

Comorbidities	No. of cases	Percentage
Type II Diabetes mellitus	30	24.4%
Hypertension	24	19.5%
Coronary Artery Disease (CAD)	6	4.9%
Cerebrovascular Accident (CVA)	2	1.6%
Chronic Kidney Disease (CKD)	7	5.7%
Thyroid disease	8	6.5%
Chronic Liver Disease (CLD)	2	1.6%
Rheumatoid Arthritis	1	0.8%
Total	80	65%

There were 80 patients (65%) presenting with severe thrombocytopenia who were associated with the comorbid conditions. Most common was type 2 Diabetes Mellitus (24.4%) followed by Hypertension (19.5%).

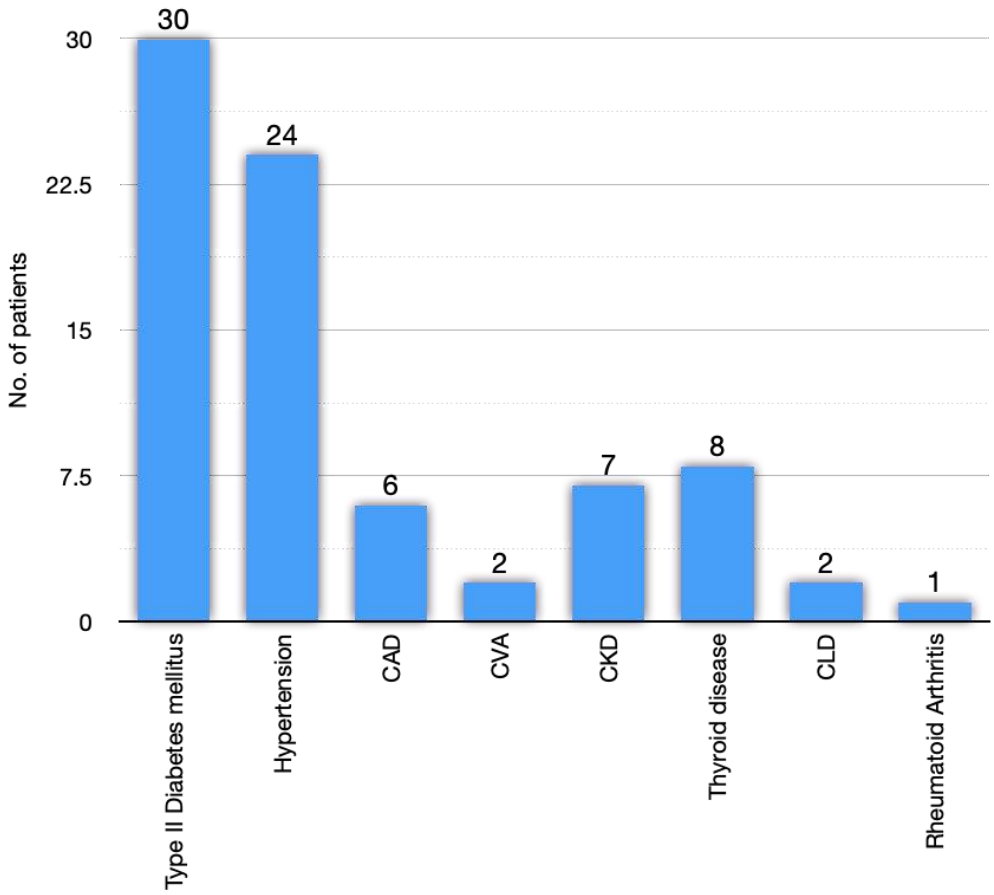

Figure 12: Bar graph showing distribution of comorbidities associated with patients presented with severe thrombocytopenia.

Table 11: Distribution of patients with severe thrombocytopenia according to symptoms

Symptoms	No. of cases	Percentage
Fever	90	73.2%
Malaise/Fatigue/Generalized body weakness	44	35.8%
Nausea	17	13.8%
Vomiting	25	20.3%
Diarrhea	09	7.3%
Constipation	02	1.6%
Breathlessness	25	20.3%
Abdominal pain	28	22.8%
Weight loss	2	1.6%

Fever was the predominant presenting complaints (73.2%). Generalized body weakness was seen in 44 patients (35.8%). Patients with abdominal pain numbered 28 (22.8%).

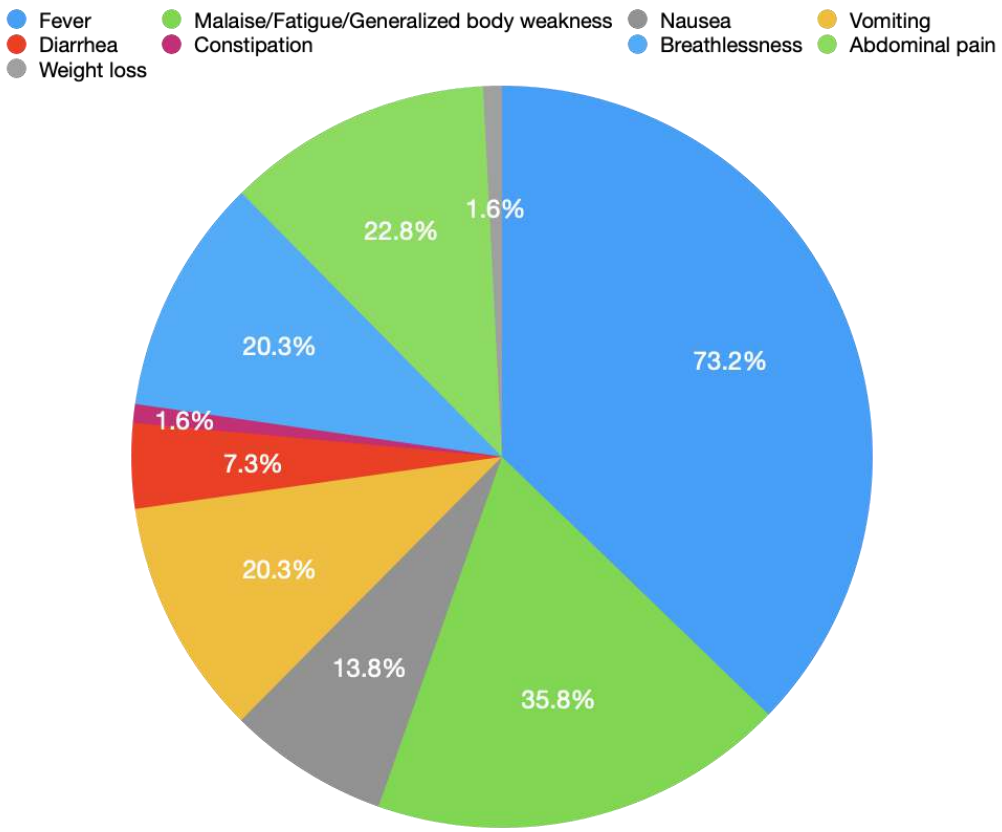

Figure 13: Pie chart showing distribution of comorbidities associated with patients presented with severe thrombocytopenia

Table 12: Incidence of bleeding events in different etiologies

	No. of cases with bleeding manifestation	Total no of patients	Percentage
Tropical fever	16	61	26.22%
Sepsis	10	23	43.47%
ITP	14	18	77.77%
Autoimmune	3	3	100.0%
Drug induced	1	1	100.0%
Megaloblastic Anaemia	0	2	0.0%
Tuberculosis	0	3	0.0%
Multifactorial	9	12	75.0%
Total	53	123	43.08%

Out of the total 123 patients, 53 patients (43.08%) had bleeding manifestation. Approximately three fourths of the total patients in ITP and Multifactorial groups showed bleeding manifestations. All the patients of Autoimmune and Drug induced thrombocytopenia showed some form of bleeding manifestations. In the sepsis group less than half of the patients (43.47%) showed these features while one fourth of the Tropical fever group showed bleeding manifestations. In patients with Tuberculosis and Megaloblastic Anaemia showed no form of bleeding manifestation.

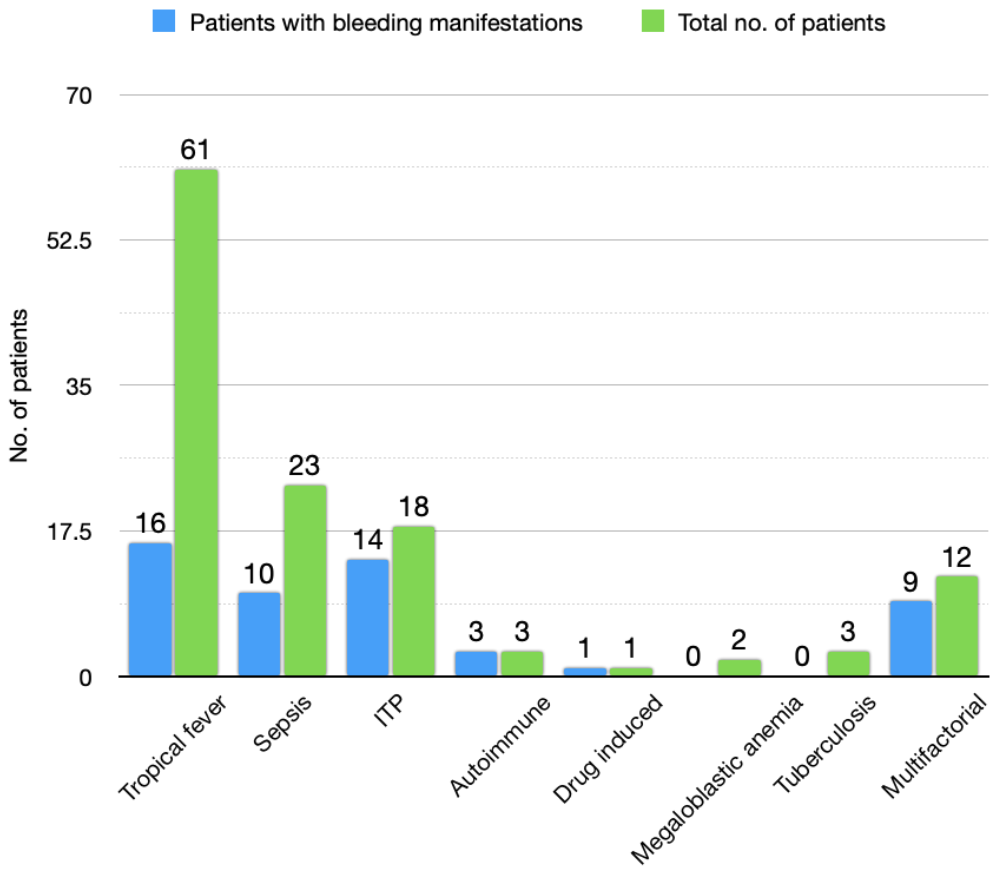

Figure 14: Bar graph showing presence of various bleeding manifestations in different etiologies of thrombocytopenia

Table 13: Bleeding manifestation seen in patients with severe thrombocytopenia

	No. of patients	Incidence
Subconjunctival Haemorrhage	3	2.44%
Epistaxis	5	4.07%
Gum Bleeding	16	13.01%
Ecchymosis	21	17.07%
Malena	16	13.01%
Hematuria	5	4.07%
Menorrhagia	6	4.88%

Patients with severe thrombocytopenia presented with various forms of bleeding manifestation. Most common form of bleeding manifestation was ecchymosis (17.07%) followed by gum bleeding (13.01%) and malena (13.01%). Other forms of bleeding manifestation included menorrhagia (4.88%), epistaxis (4.07%), hematuria (4.07%) and subconjunctival Haemorrhage (2.44%).

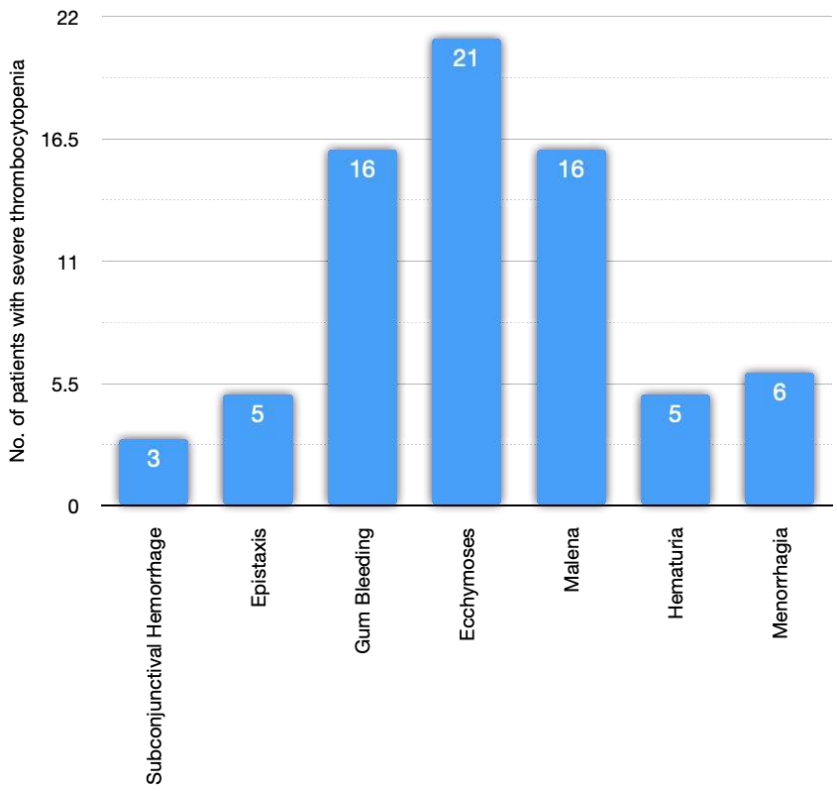

Figure 15: Bar graph showing bleeding manifestation seen in patients with severe thrombocytopenia

Table 14: Bleeding manifestations seen in Tropical fever patients

	No. of patients	Incidence
Subconjunctival Haemorrhage	0	0.00%
Epistaxis	1	1.64%
Gum Bleeding	5	8.20%
Ecchymosis	3	4.92%
Malena	6	9.84%
Hematuria	2	3.28%
Menorrhagia	2	3.28%

16 out of the 61 patients had bleeding manifestation in tropical fever. Most common bleeding manifestation seen in tropical fever was malena (9.84%) followed by gum bleeding (8.20%).

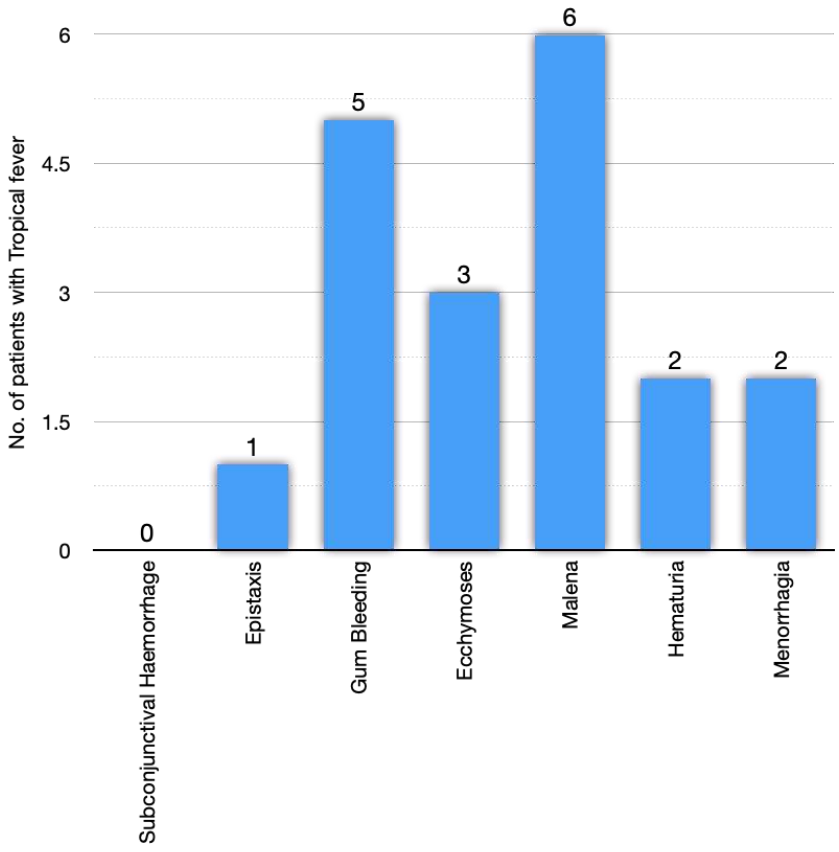

Figure 16: Bar graph showing distribution of bleeding manifestations seen in patients with Tropical fever

Table 15: Bleeding manifestations seen in patients with Sepsis

	No. of patients	Incidence
Subconjunctival Haemorrhage	1	4.35%
Epistaxis	0	0.00%
Gum Bleeding	1	4.35%
Ecchymosis	6	26.09%
Malena	3	13.04%
Hematuria	0	0.00%
Menorrhagia	0	0.00%

10 out of 23 patients had bleeding events in sepsis. Most common bleeding manifestation seen in sepsis was ecchymosis (26.09%).

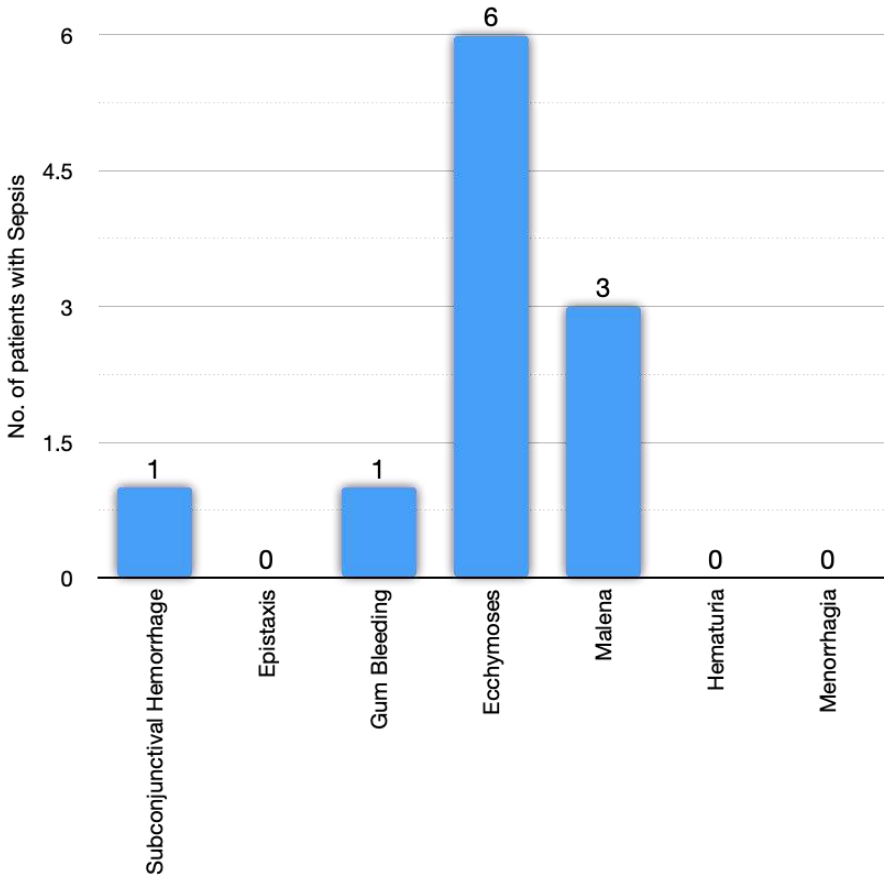

Figure 17: Bar graph showing distribution of bleeding manifestations seen in patients with Sepsis

Table 16: Bleeding manifestations seen in patients with ITP

	No. of patients	Incidence
Subconjunctival Haemorrhage	1	5.56%
Epistaxis	4	22.22%
Gum Bleeding	7	38.89%
Ecchymosis	5	27.78%
Malena	4	22.22%
Hematuria	2	11.11%
Menorrhagia	2	11.11%

14 out of the 18 patients with ITP had bleeding events. Most common bleeding event observed was gum bleeding (38.89%). Other forms of bleeding manifestation in descending order of frequency included ecchymosis (27.78%), epistaxis (22.22%), malena (22.22%), hematuria (11.11%), menorrhagia (11.11%) and subconjunctival Haemorrhage (5.56%).

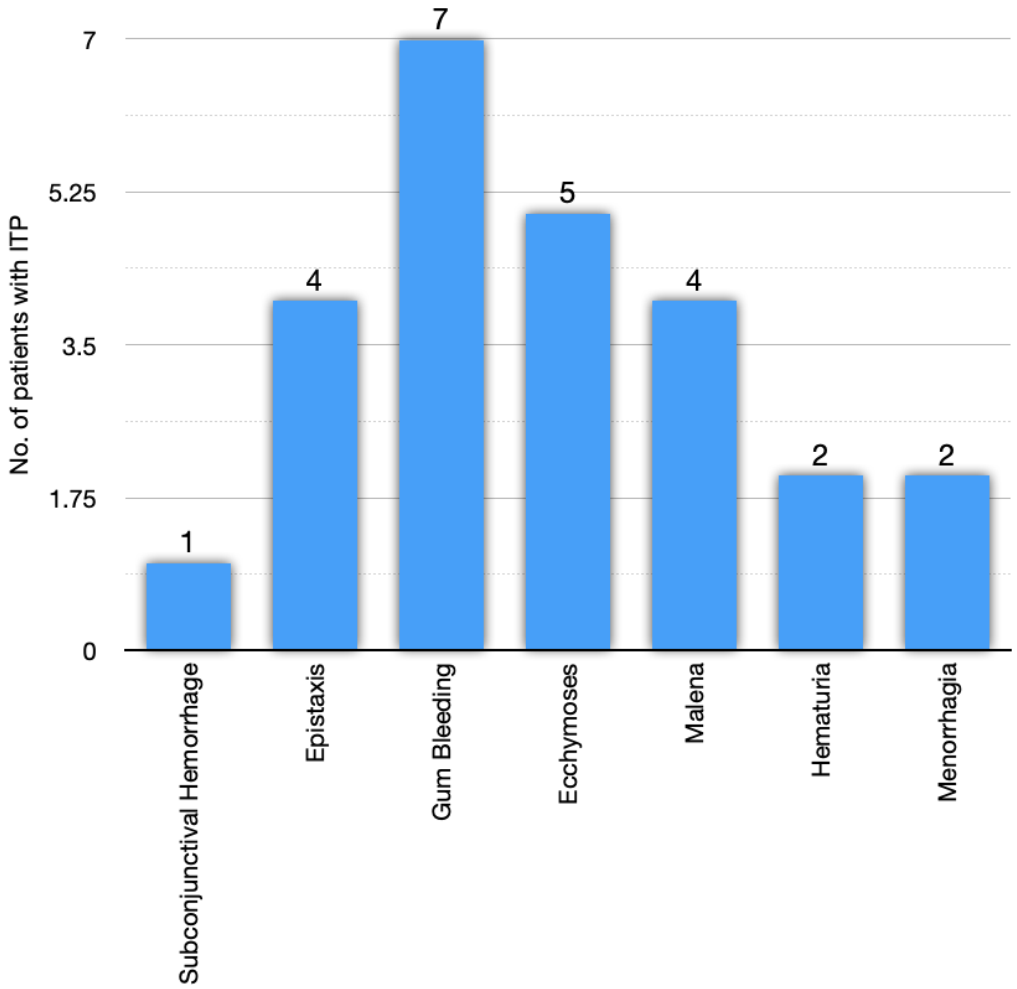

Figure 18: Bar graph showing distribution of bleeding manifestations seen in patients with ITP

Table 17: Bleeding manifestations seen in patients with Multifactorial Etiologies

	No. of patients	Incidence
Subconjunctival Haemorrhage	1	8.33%
Epistaxis	0	0.00%
Gum Bleeding	3	25.00%
Ecchymosis	6	50.00%
Malena	0	0.00%
Hematuria	1	8.33%
Menorrhagia	1	8.33%

9 out of 12 patients had bleeding manifestation in patients having thrombocytopenia attributed to the multiple etiologies. Most common form was ecchymosis which was seen in half of the total patients having multiple etiologies.

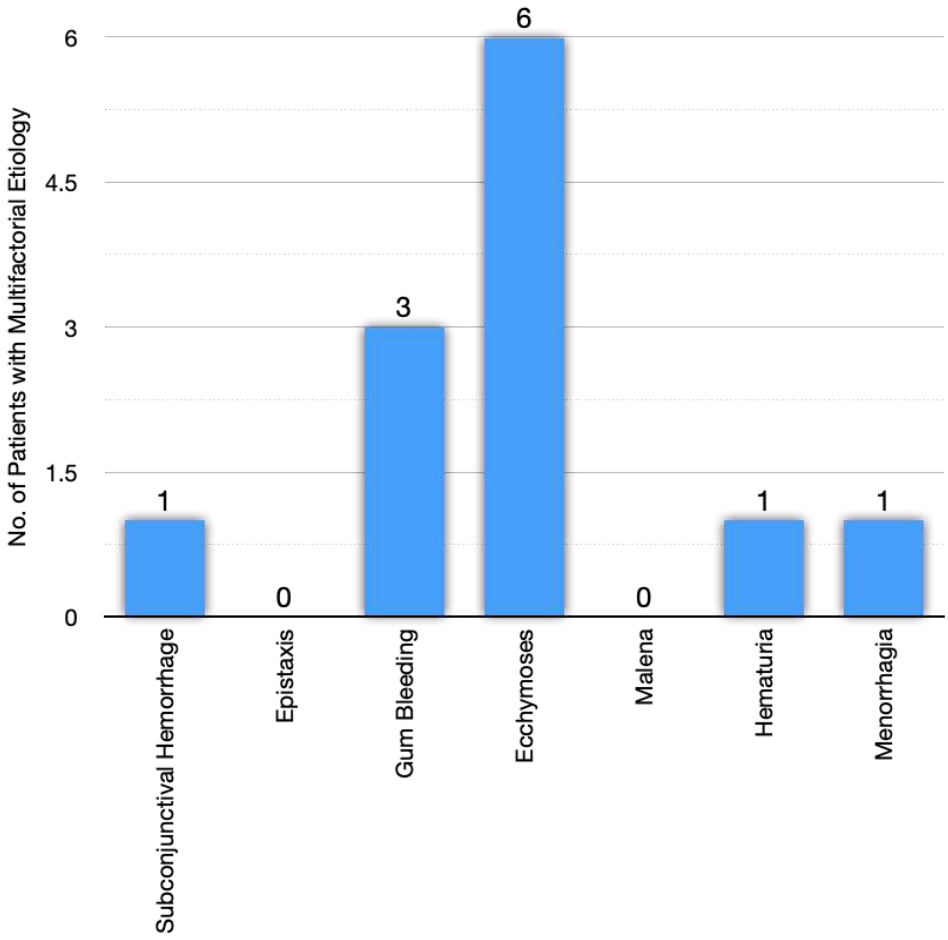

Figure 19: Bar graph showing distribution of bleeding manifestations seen in patients with Multifactorial etiologies

Table 18: Bleeding manifestations seen in patients with autoimmune cause of Thrombocytopenia

	No. of patients	Incidence
Subconjunctival Haemorrhage	0	0.00%
Epistaxis	0	0.00%
Gum Bleeding	0	0.00%
Ecchymosis	1	33.33%
Malena	2	66.67%
Hematuria	0	0.00%
Menorrhagia	1	33.33%

All the three patients having severe thrombocytopenia due to autoimmune causes had bleeding events. One patient had Malena and menorrhagia together. Malena was seen in two third of the cases whereas ecchymosis and hematuria was seen in one third of the cases each.

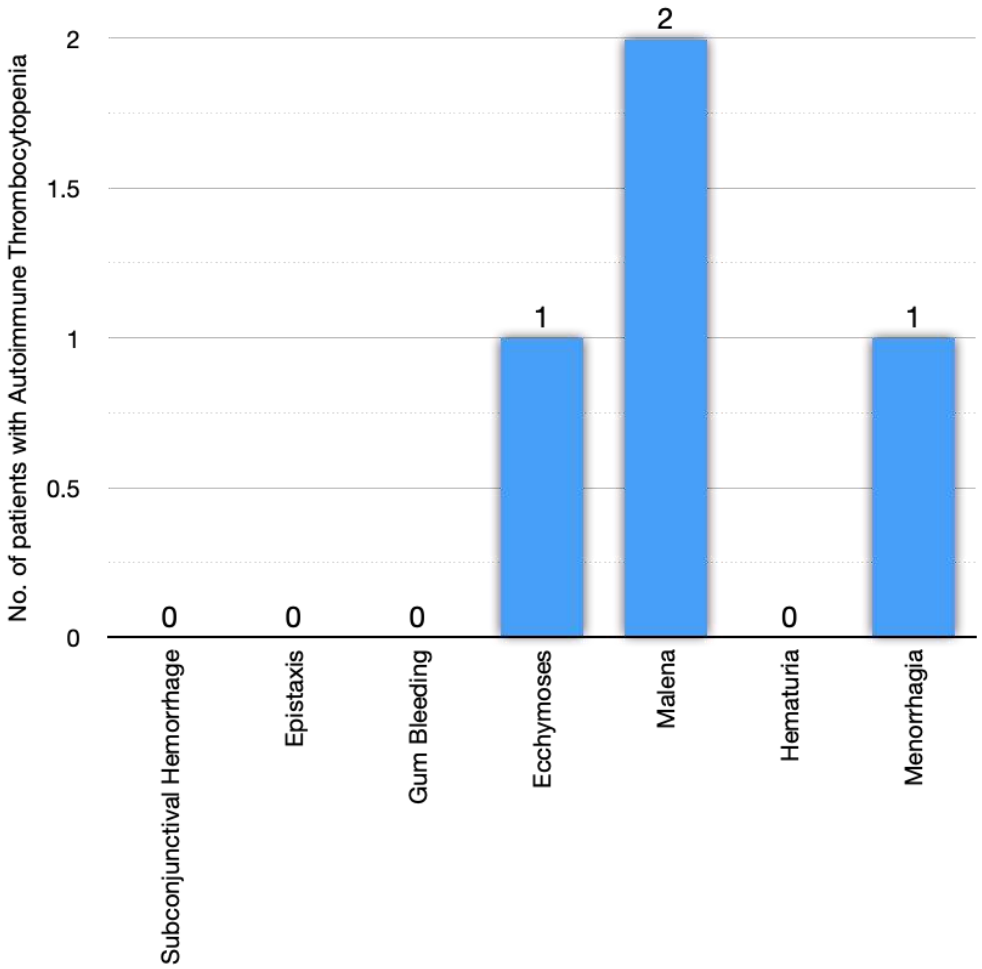

Figure 20: Bar graph showing distribution of bleeding manifestations seen in patients with Autoimmune cause of Thrombocytopenia

Table 19: Bleeding manifestations seen in patients with Drug induced Thrombocytopenia

	No. of patients	Incidence
Subconjunctival Haemorrhage	0	0.00%
Epistaxis	0	0.00%
Gum Bleeding	0	0.00%
Ecchymosis	0	0.00%
Malena	1	100.00%
Hematuria	0	0.00%
Menorrhagia	0	0.00%

The one patient with drug induced severe thrombocytopenia had malena. Patients with Thrombocytopenia due to Tuberculosis and Megaloblastic Anaemia did not present with any bleeding manifestation.

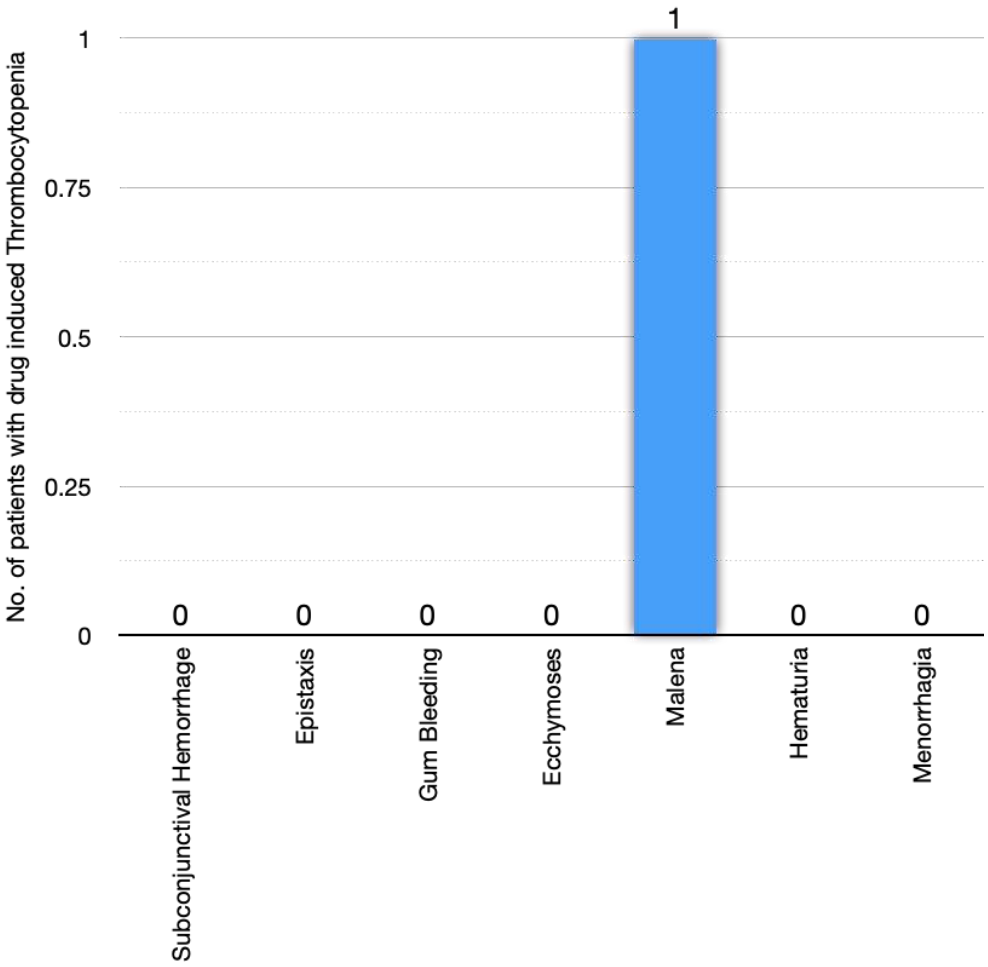

Figure 21: Bar graph showing distribution of bleeding manifestations seen in patients with drug induced Thrombocytopenia

Table 20 Distribution of patients according to signs

	No. of cases	Percentage
Petechiae	20	16.3%
Ecchymosis	21	17.1%
Hepatomegaly	09	7.3%
Splenomegaly	17	13.8%
Lymphadenopathy	0	0.0%
Total	67	54.5%

21 (17.1%) patients presented with ecchymosis at admission. Petechiae was noted in 20 patients (16.3%). None of the patients had any significant lymphadenopathy. Hepatomegaly and splenomegaly seen in 9 patients (7.3%) and 17 patients (13.8%) respectively.

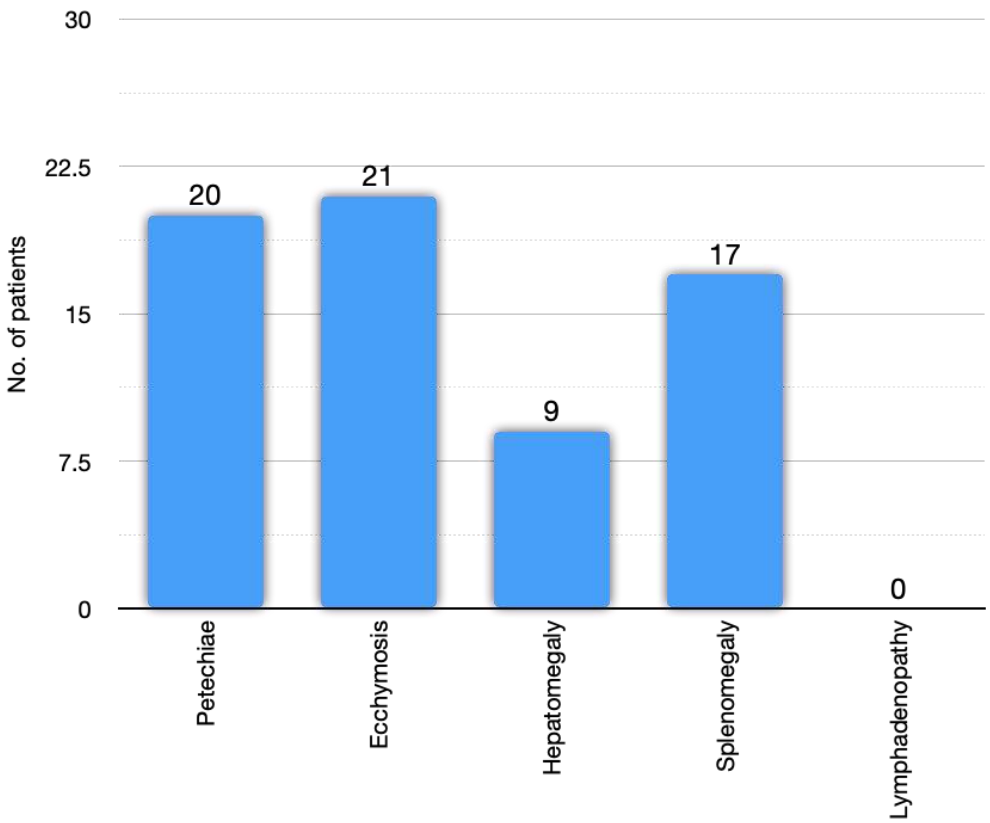

Figure 22: Bar graph showing distribution of patients according to signs

Table 21: No. of patients with bone marrow examination.

Bone marrow(yes/no)	No. of cases	Percentage
Yes	40	32.52%
No	83	67.47%
Total	123	100%

Bone marrow was done in 40 patients (32.52%)

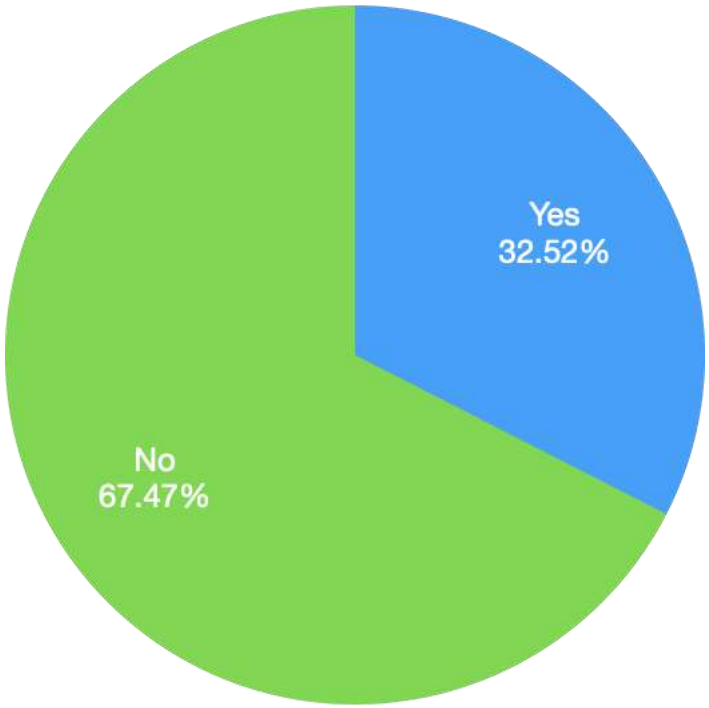

Figure 23: Pie chart showing distribution of patients in whom Bone Marrow examination was done

Table 22: No. of patients with various etiologies in whom bone marrow examination was done

Etiology	No. of patients	Percentage
Tropical fever	2	5.00%
Sepsis	4	10.00%
ITP	18	45.00%
Tuberculosis	2	5.00%
Autoimmune	3	7.50%
Megaloblastic anaemia	1	2.50%
Drug induced	0	0.00%
Multifactorial	10	25.00%
Total	40	100.00%

All the patients with ITP had their bone marrow examination done. Out of total bone marrow examination, 45% were done in ITP patients. One fourth of the total bone marrow was done in patients with thrombocytopenia due to multiple etiologies. 10% of the bone marrow was done in sepsis, 7.5% in autoimmune causes, 5.00% in tropical fever and tuberculosis each. 2.5% of the bone marrow was done in megaloblastic anaemia. Bone marrow was not done in the patient with drug induced severe thrombocytopenia.

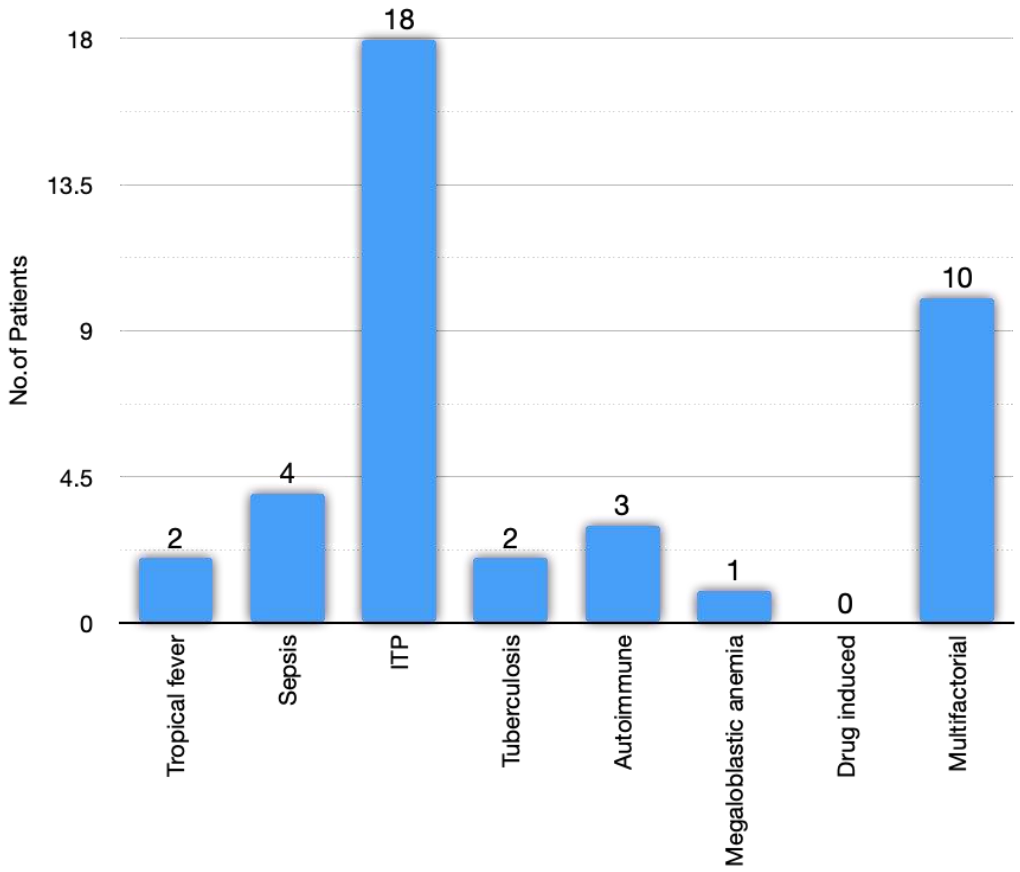

Figure 24: Bar graph showing distribution of patients with various etiologies in whom bone marrow examination was done

Table 23: Distribution of Bone Marrow Cellularity in patients with Thrombocytopenia

Marrow Cellularity	No. of cases	Percentage
Hypercellular	26	65.00%
Normocellular	14	35.00%
Hypocellular	0	0.0%
Total	40	100.0%

Out of the 40 patients, 26 patients (65.00%) showed hypercellular marrow and 14 patients (35.00%) had normocellular marrow. Hypocellular marrow was not seen in any of our patients.

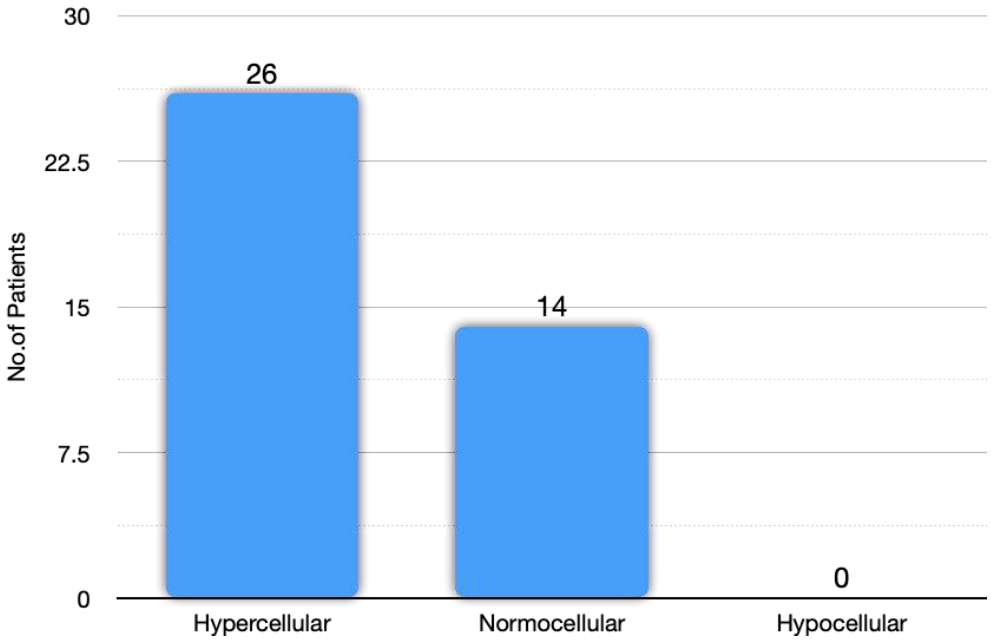

Figure 25: Bar graph showing distribution of Bone Marrow Cellularity in patients with Thrombocytopenia

Table 24: Distribution of Bone Marrow Cellularity findings as per various etiologies of Thrombocytopenia

Etiology	Hypercellular	Percentage	Normocellular	Percentage
Autoimmune	2	7.69%	1	7.14%
ITP	12	46.15%	6	42.86%
Drug induced	0	0.00%	0	0.00%
Tuberculosis	0	0.00%	2	14.29%
Tropical fever	2	7.69%	0	0.00%
Sepsis	2	7.69%	2	14.29%
Megaloblastic Anaemia	1	3.85%	0	0.00%
Multifactorial	7	26.92%	3	21.43%
Total	26	100.00%	14	100.00%

Among the 18 ITP patients, 12 patients had hypercellular marrow and 6 patients had normocellular marrow.

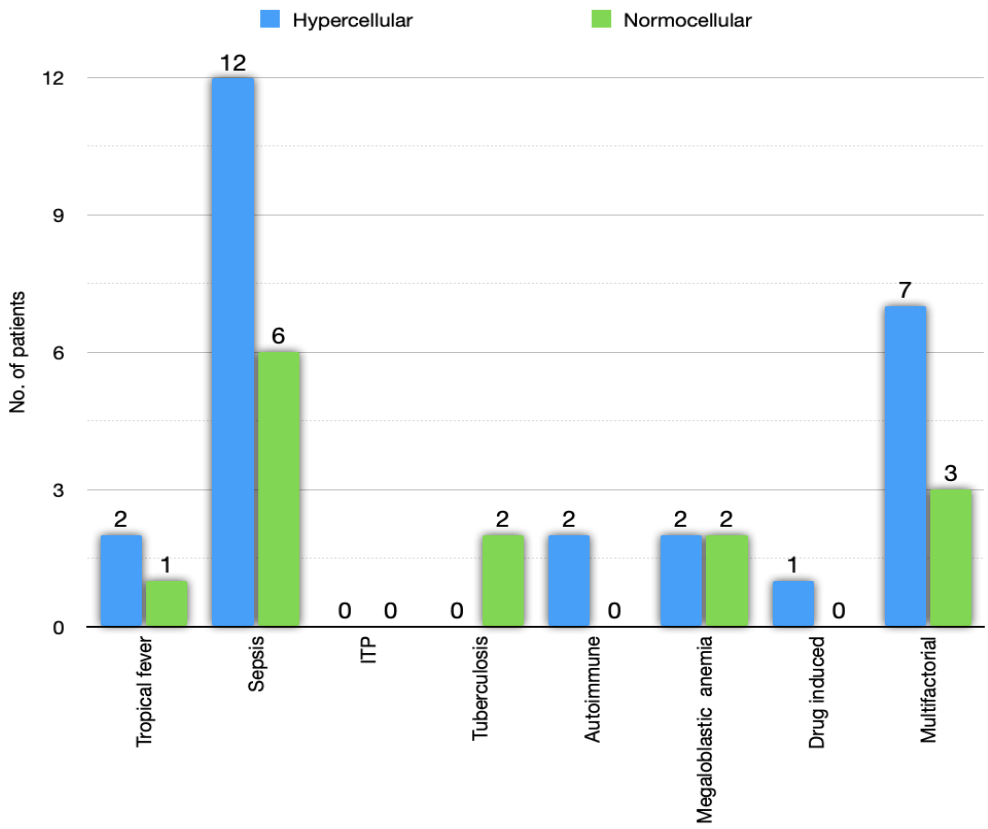

Figure 26: Bar graph showing distribution of Bone Marrow Cellularity findings as per various etiologies of Thrombocytopenia

Table 25: Distribution of patients according to the other bone marrow findings in patients in whom bone marrow examination was done

Findings of Bone marrow	No. of cases	Percentage
Erythroid Prominence	5	12.50%
Granulomatous Inflammation	2	5.00%
Hemophagocytosis	4	10.00%
ITP	25	62.50%
ITP with IDA	2	5.00%
Megaloblastic Anaemia	1	2.50%
Normal	1	2.50%

Out of 40 bone marrows, there were 25 (62.50%) bone marrows suggesting hyper-destructive thrombocytopenia. 4 patients (10.00%) showed Hemophagocytosis in bone marrow and 5 patients had erythroid prominence (12.5%) in bone marrow. Granulomatous inflammation was seen in 2 patients (5.00%) in marrow.

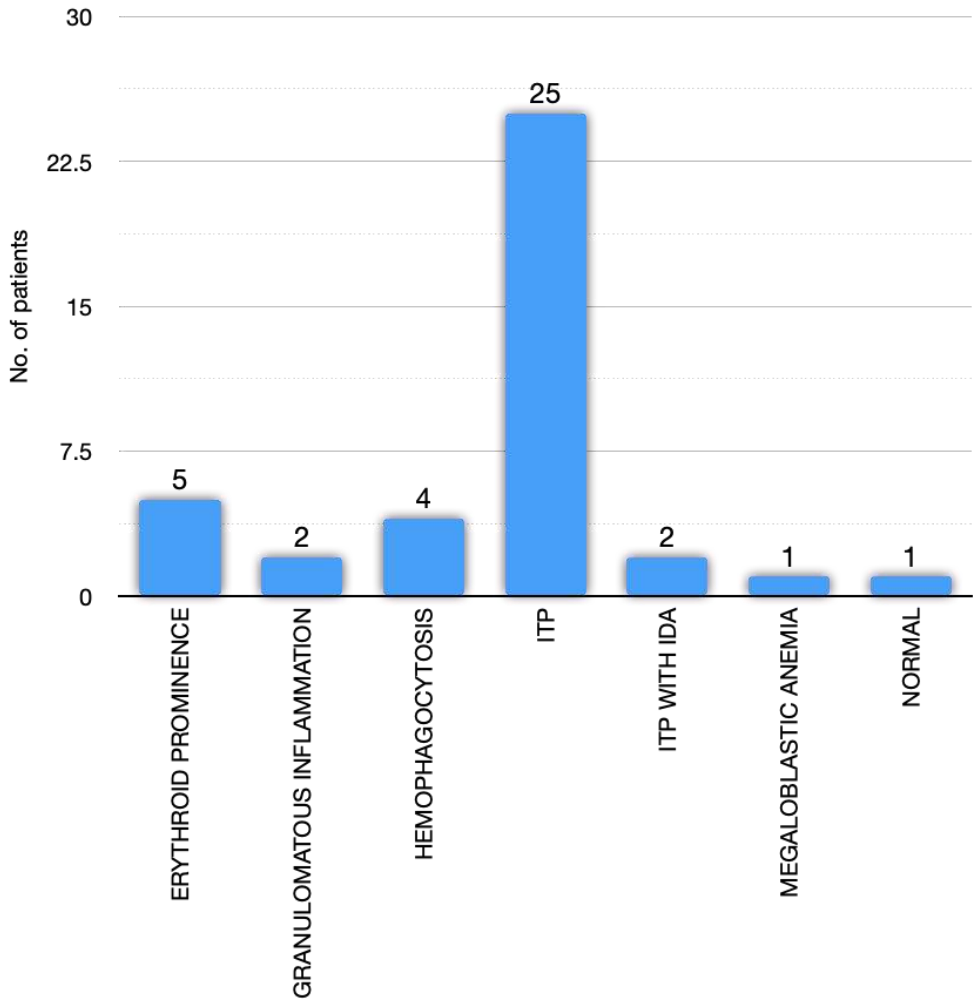

Figure 27: Bar graph showing distribution of patients according to the other bone marrow findings in patients in whom bone marrow examination was done

Table 26: Distribution of patients with various etiologies as per bone marrow findings

	No. of patients in whom bone marrow done	Bone marrow findings		
		Finding	No. of patients	Percentage
Tropical fever	2	Erythroid prominence	2	100.00%
Sepsis	4	Erythroid prominence	3	75.00%
		Normal	1	25.00%
ITP	18	ITP	17	94.44%
		ITP With IDA	1	5.55%
Tuberculosis	2	Granulomatous Inflammation	2	100.0%
Autoimmune	3	ITP	2	66.7%
		ITP With IDA	1	33.3%
Megaloblastic Anaemia	1	Megaloblastic Anaemia	1	100.0%
Drug induced	0	-	-	-
Multifactorial	10	Hemophagocytosis	4	40.00%
		ITP	6	60.00%

Among tropical fever, there are 2 patients in whom bone marrow was done, both bone marrow suggested erythroid prominence. There were 4 patients with sepsis induced severe thrombocytopenia in whom bone marrow examination was done. Three fourth of them showed erythroid prominence and one fourth showed normal marrow. Bone marrow examination was done in all the ITP patients, 94.44% of them showed hyper destructive thrombocytopenia and 5.55% showed hyper

destructive thrombocytopenia with iron deficiency Anaemia. Bone marrow examination done in the two patients having thrombocytopenia due to tuberculosis suggested granulomatous inflammation. There were 3 patients with severe thrombocytopenia due to autoimmune causes, bone marrow was done in all of them. 66.7% showed hyper destructive thrombocytopenia and 33.3% showed hyper destructive thrombocytopenia with iron deficiency Anaemia. Bone marrow done in patient with megaloblastic Anaemia suggested features of megaloblastic Anaemia in the bone marrow. Among the 12 patients with thrombocytopenia due to multifactorial etiology, bone marrow was done in 10 patients. 4 out of 10 had Hemophagocytosis in the bone marrow and rest of the 6 patients bone marrow suggested hyper destructive thrombocytopenia. Bone marrow examination was not done in patient with drug induced severe thrombocytopenia.

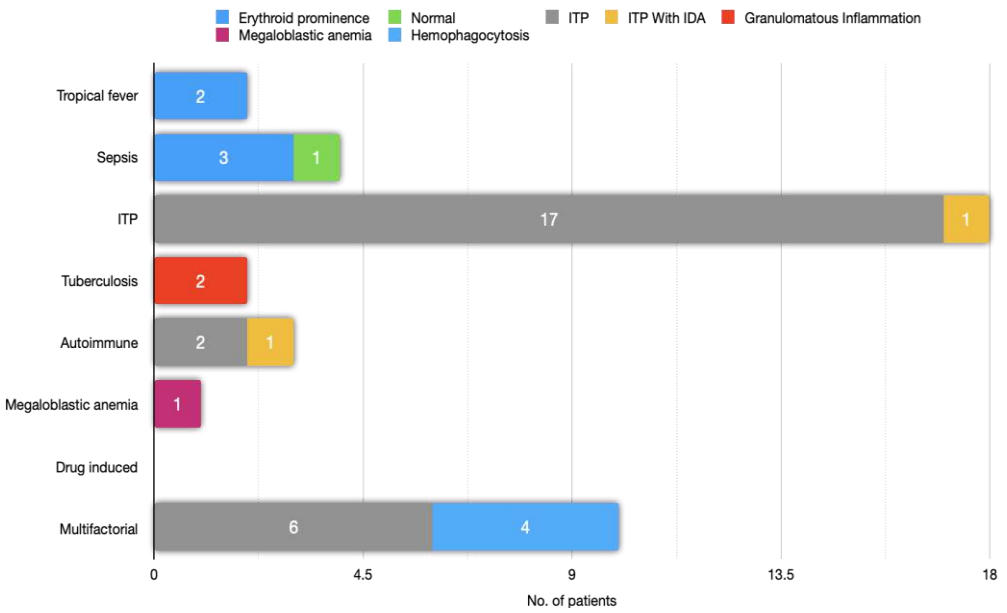

Figure 28: Bar graph showing distribution of patients with various etiologies as per bone marrow findings

Table 27: Distribution of patients according to the ANA Immunofluorescence (ANA-IF)

ANA-IF	No. of cases	Percentage
1+	6	16.2%
2+	4	10.8%
3+	5	13.5%
4+	2	5.4%
Negative	20	54.1%
Total	37	100.0%

Autoimmune work up was done in 37 patients and was negative in 20 patients (54.1%). Out of the 17 patients with positive results, ANA-IF 3+ and 4+ results were seen in 5 patients (13.5%) and 2 patients (5.4%) respectively. ANA profile was done in patients with positive ANA-IF.

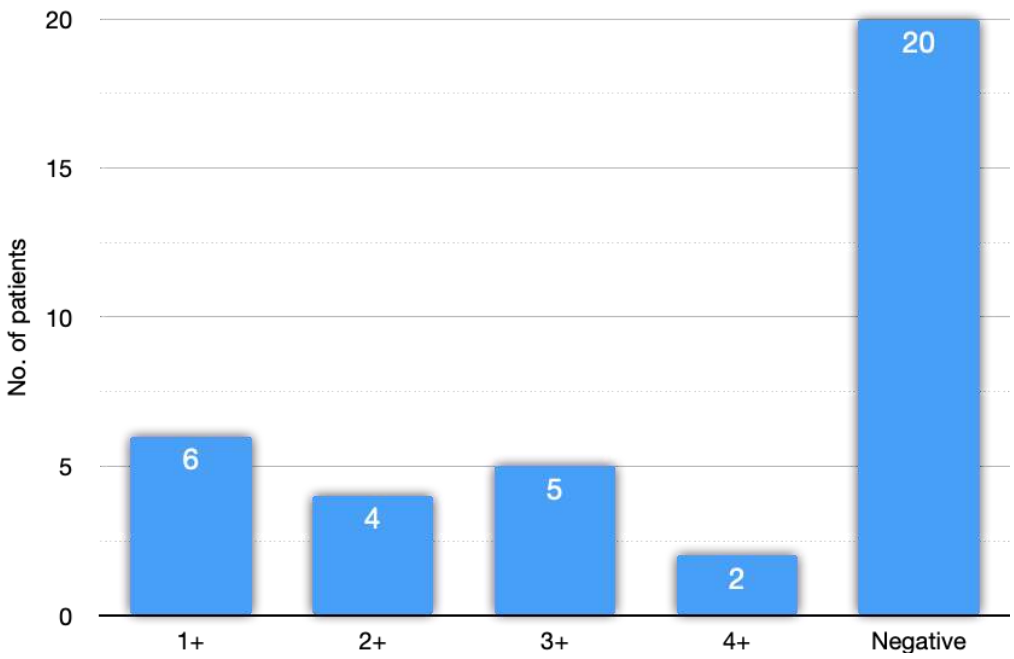

Figure 29: Bar graph showing distribution of patients according to the results of ANA Immunofluorescence (ANA-IF)

Table 28: Results of ANA profile in patients with positive ANA-IF

ANA Profile and APLA	No. of cases	Percentage
Negative	10	8.1%
Ku/ uctd	1	0.8%
SSA/Ro52 , SSB/LA	4	3.3%
DsDNA	1	0.8%
Lupus anticoagulant	1	0.8%

There was total 17 patients with positive ANA IF. Out of these 17 patients, 10 patients had negative ANA profile .4 patients had positive SSA/Ro52 antibody (seen in Sjogren syndrome). Other antibodies seen in the patients included Ku/ uctd (seen in undifferentiated connective tissue disorder), DsDNA (seen in SLE) and Lupus anticoagulant (seen in APLA syndrome).

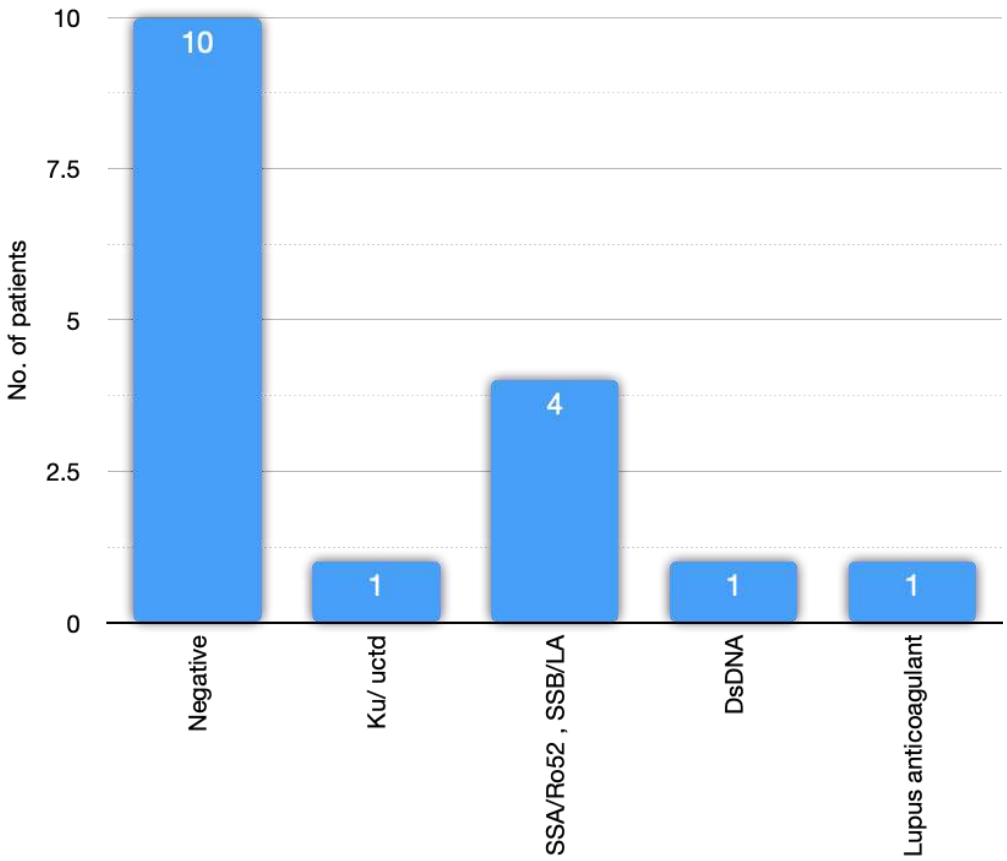

Figure 30: Bar graph showing distribution of patients based on the results of ANA profile in patients with positive ANA-IF

Table 29: Distribution of patients according to Hemoglobin (Hb) levels

Hb levels (in gm/dL)	No. of patients	Percentage
<10	30	24.39%
10-13	37	30.08%
>13	56	45.53%
Total	123	100.00%

One fourth of the total number of patients (n=30, 24.39%) presented with Hb levels <10 gm/dL. 30.08% patients had Hb between 10 -13 gm/dl and 45.53% patients had Hb more than 13 gm/dl.

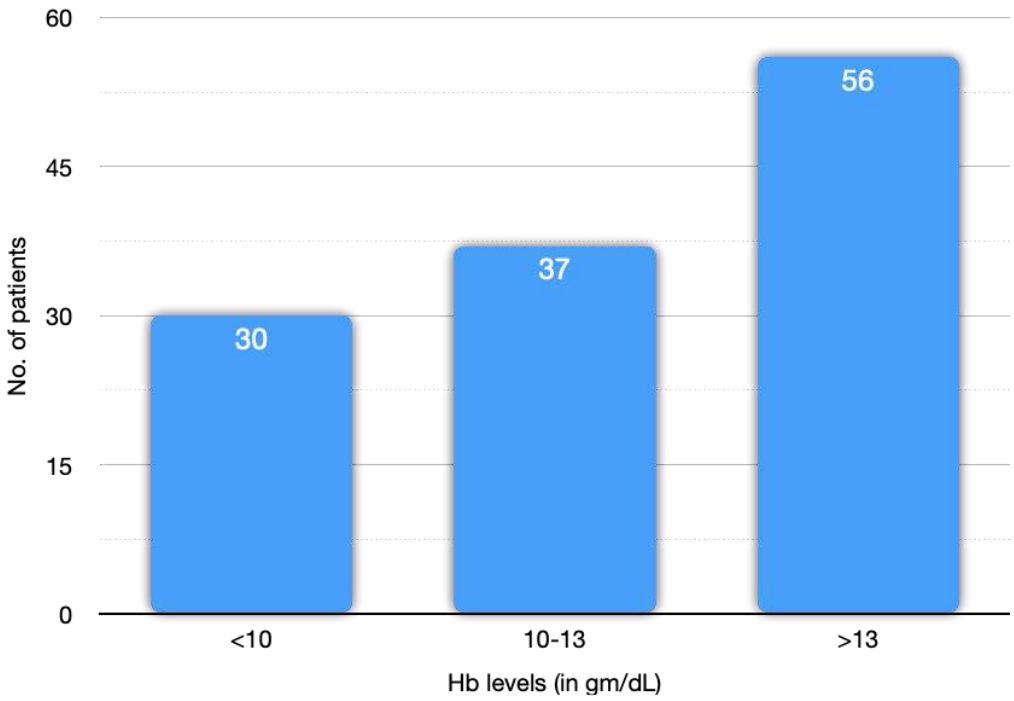

Figure 31: Bar Graph showing distribution of patients according to Hemoglobin (Hb) levels

Table 30: Peripheral Blood Film (PBF) findings in the patients with severe thrombocytopenia

PBF findings	No. of patients	Percentage
Normocytes	97	78.9%
Microcytes	13	10.6%
Macrocytes	7	5.7%
Toxic granules	6	4.9%
Total	123	100.0%

Most of the patients with severe thrombocytopenia had normocytes (78.9%) in the peripheral blood film followed by microcytes (10.6%). 6 patients (4.9%) showed toxic granules in peripheral blood film.

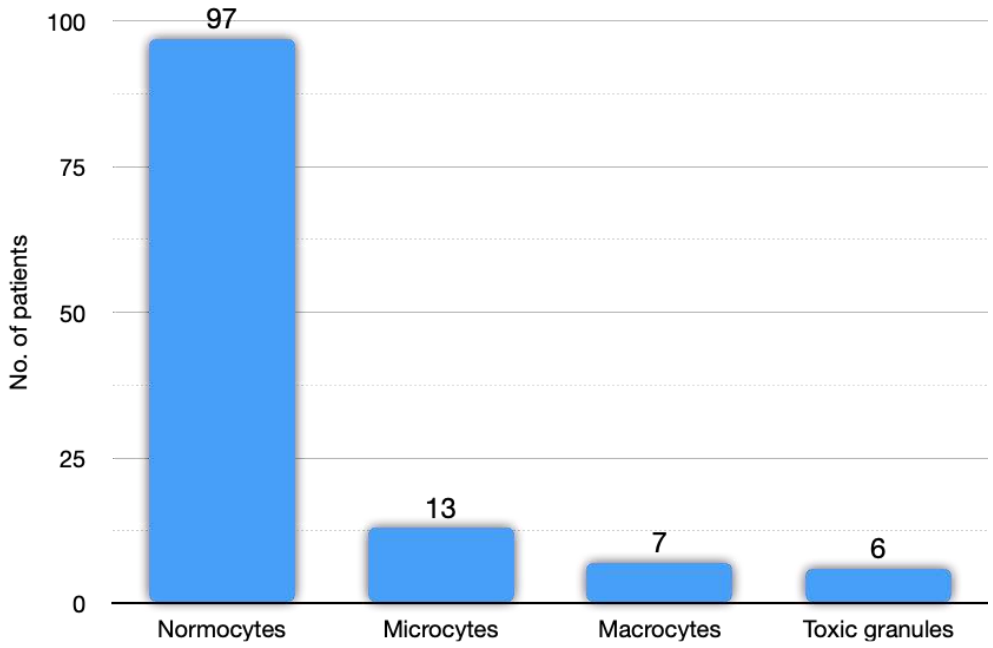

Figure 32: Bar graph showing distribution of patients based on Peripheral Blood Film (PBF) findings in the patients with severe thrombocytopenia

Table 31: Distribution of patients according to TLC values

TLC (counts/ µL)	No. of cases	Percentage
< 4000	26	21.1%
4000-11000	71	57.7%
> 11000	26	21.1%
Total	123	100.0%

26 patients (21.1%) had TLC count <4000/ µL. Out of the 123 enrolled patients, 71 (57.7%) patients had TLC counts between 4000 - 11000/µL.

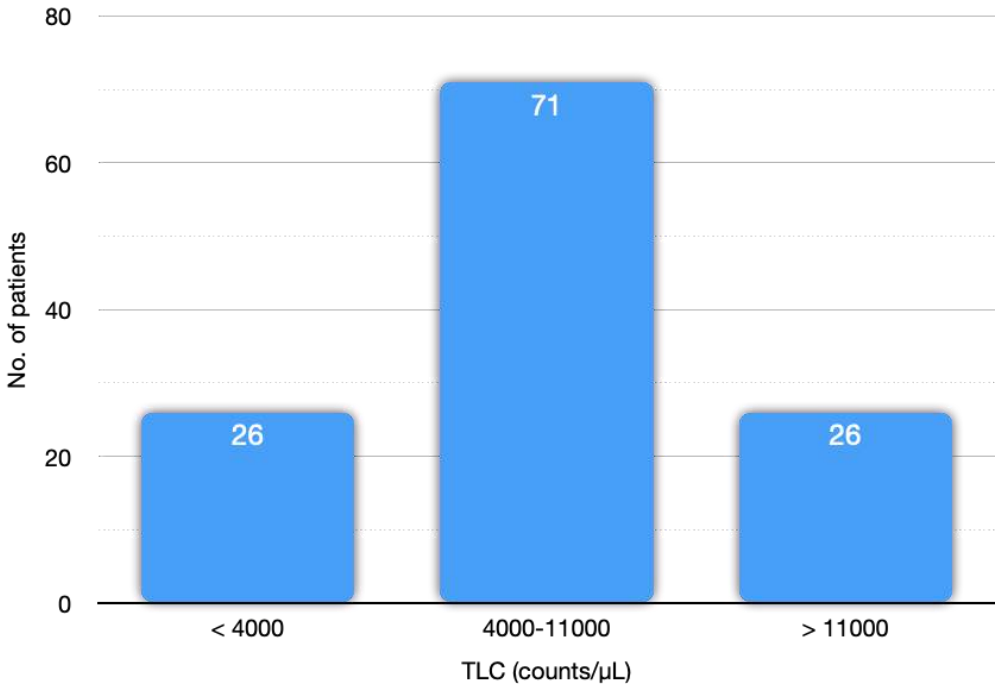

Figure 33: Bar graph showing Distribution of patients according to TLC values

Table 32: Distribution of patients with Pancytopenia and Bicytopenia in severe thrombocytopenia

	No. of patients	Percentage
Pancytopenia	12	9.76%
Bicytopenia	32	26.02%
Isolated Thrombocytopenia	79	64.23%
Total	123	100.00%

Approximately two thirds of total number of patients (n=79, 64.23%) presented with isolated Thrombocytopenia. Only 12 patients (9.76%) presented with Pancytopenia (Hb <10.00 gm/dL, TLC <4000/ µL).

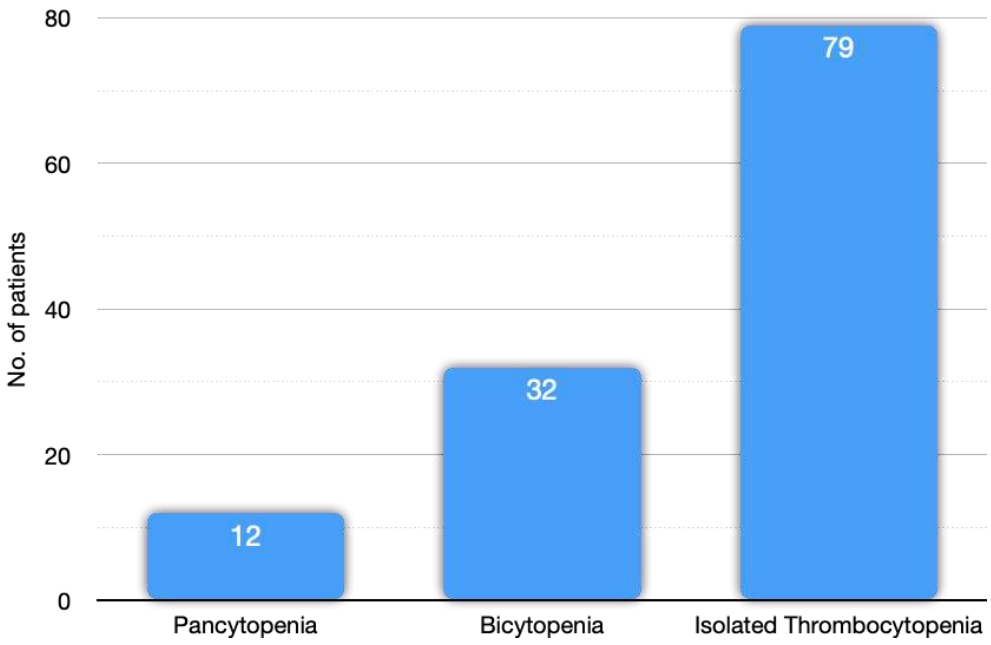

Figure 34: Distribution of patients with Pancytopenia and Bicytopenia in severe thrombocytopenia

Table 33: Bone marrow findings in patients with Pancytopenia

	No. of patients	Percentage
Erythroid Prominence	2	28.57%
Granulomatous Inflammation	0	0.00%
Hemophagocytosis	1	14.29%
ITP	2	28.57%
ITP with IDA	2	28.57%
Megaloblastic Anaemia	0	0.00%
Normal	0	0.00%
Total	7	100.00%

Out of 12 patients with pancytopenia, bone marrow examination was done in 7 patients. Erythroid prominence, ITP, ITP with IDA were the most common findings in the bone marrow examination of patients with pancytopenia in our study.

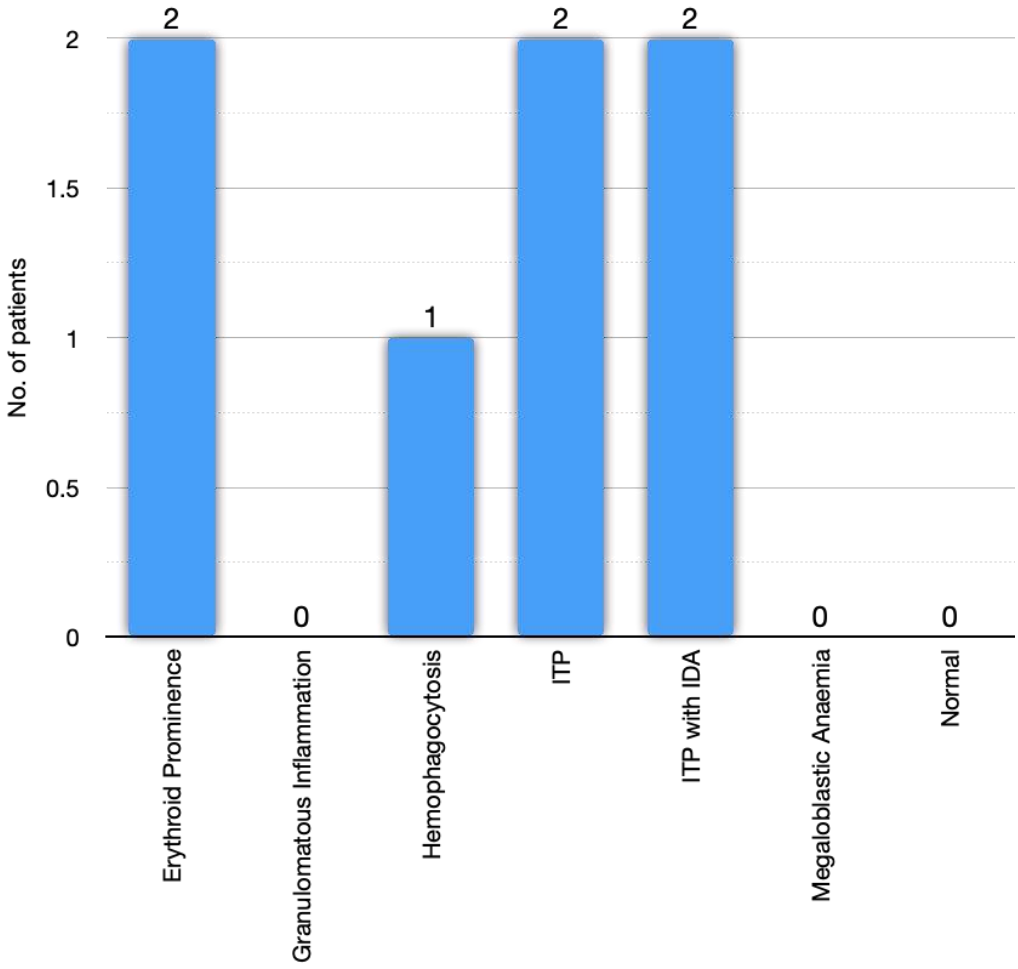

Figure 35: Bar graph showing distribution Bone marrow findings in patients with Pancytopenia

Table 34: Bone marrow findings in patients with Bicytopenia

	No. of patients	Percentage
Erythroid Prominence	2	20.00%
Granulomatous Inflammation	1	10.00%
Hemophagocytosis	3	30.00%
ITP	3	30.00%
ITP with IDA	0	0.00%
Megaloblastic Anaemia	1	10.00%
Normal	0	0.00%
Total	10	100.00%

Out of 32 patients with bicytopenia, bone marrow examination was done in 10 patients. Majority of the findings in bone marrows included Hemophagocytosis (30%) and ITP (30%). Erythroid prominence (20%) was the next most common finding in the bone marrow examination.

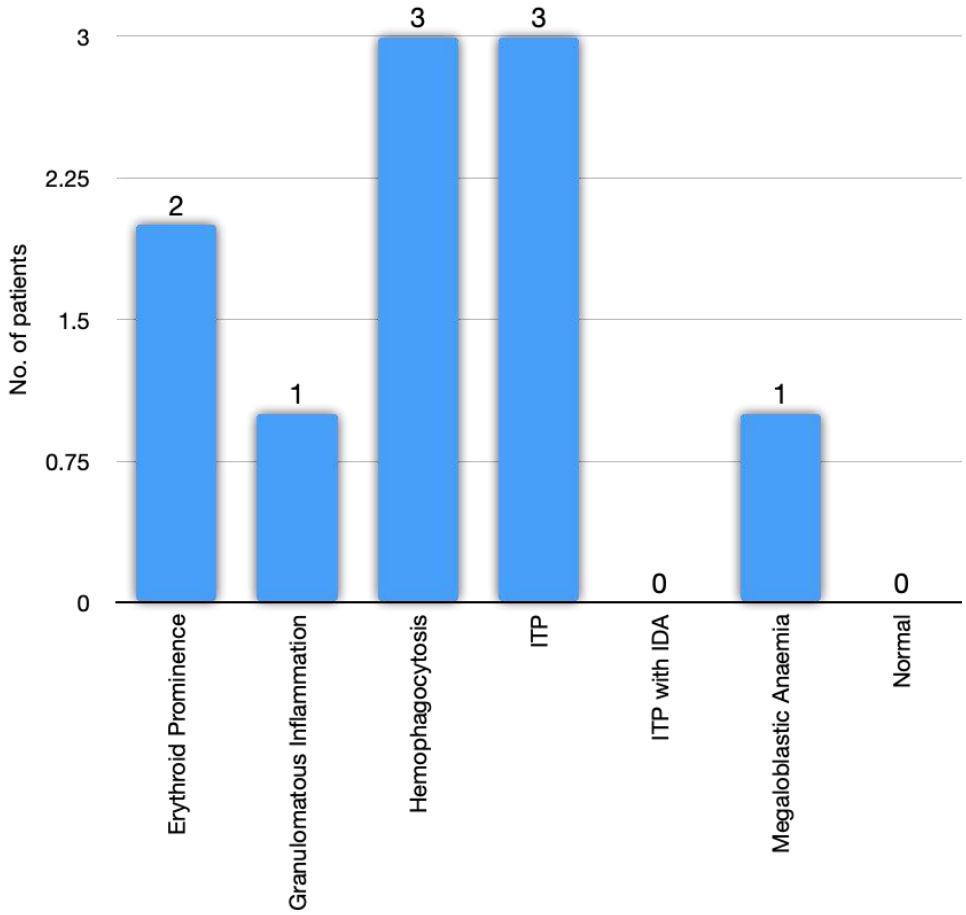

Figure 36: Bar graph showing distribution of bone marrow findings in patients with Bicytopenia

Table 35: Different treatment modalities used in study in our study we used the following treatment modalities

Treatment Modalities	No. of cases	Percentage
TPO-RA	4	3.25%
IVIG	3	2.44%
Steroids	38	30.89%
RDP, SDP	100	81.30%

Blood products were transfused in 81.3% of the patients (100 patients). Steroids were given in 38 patients (30.89%) with severe thrombocytopenia. Thrombopoietin Receptor Agonists (TPO-RA) were received by 3.25% patients (4 patients). Only 2.44% patients were given IVIG.

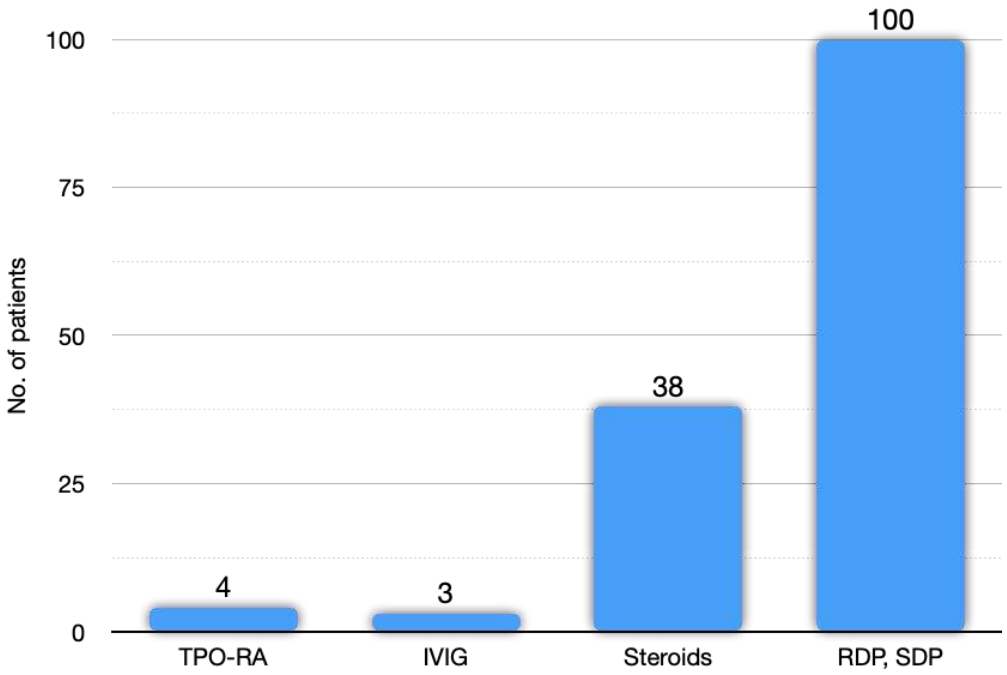

Figure 37: Bar graph showing distribution of different treatment modalities used in study in our study we used the following treatment modalities

Table 36: Distribution of patients with severe thrombocytopenia with different etiologies receiving transfusion (RDP/SDPs)

	No. of patients transfusion was given	Total no. of patients with that etiology	Percentage
Tropical Fever	55	61	90.16%
Autoimmune	3	3	100.00%
ITP	11	18	61.11%
Drug induced	0	1	0.00%
Megaloblastic Anaemia	2	2	100.00%
Sepsis	19	23	82.61%
Multifactorial	8	12	66.67%
Tuberculosis	2	3	66.67%
	100	123	81.30%

100 out of 123 patients (81.30%) received transfusions in the form of RDP/SDP. This table shows the percentage of of patients with different etiologies that required symptomatic treatment in the form of transfusions. All the patients with Thrombocytopenia due to Megaloblastic Anaemia and Autoimmune Thrombocytopenia required transfusion. Majority of patients with Thrombocytopenia due to Sepsis (82.61%) and Tropical fever (90.16%) required transfusions. Approximately one third of patients with Thrombocytopenia due to ITP, Tuberculosis and Multifactorial causes needed RDP/SDPs.

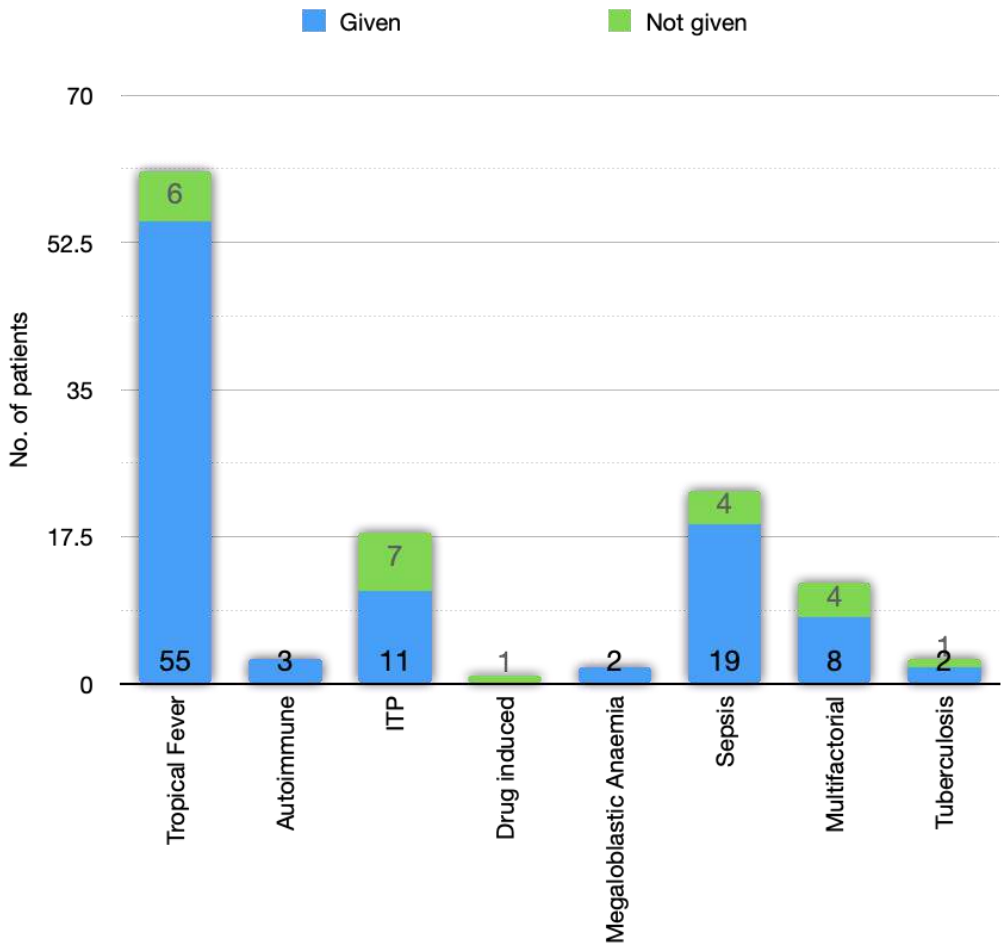

Figure 38: Bar graph showing distribution of patients with severe thrombocytopenia with different etiologies receiving transfusion (RDP/SDPs)

Table 37: Distribution of response to various treatment modalities in patients with Thrombocytopenia due to Tropical Fever

	Symptomatic Treatment	Percentage
Complete	49	80.32%
Partial Response	12	19.67%
No response	0	0.00%
Total	61	

All of the patients with Thrombocytopenia due to Tropical fever were given only symptomatic treatment. Majority of the patients (80.32%) showed complete response. None of the patients showed No response.

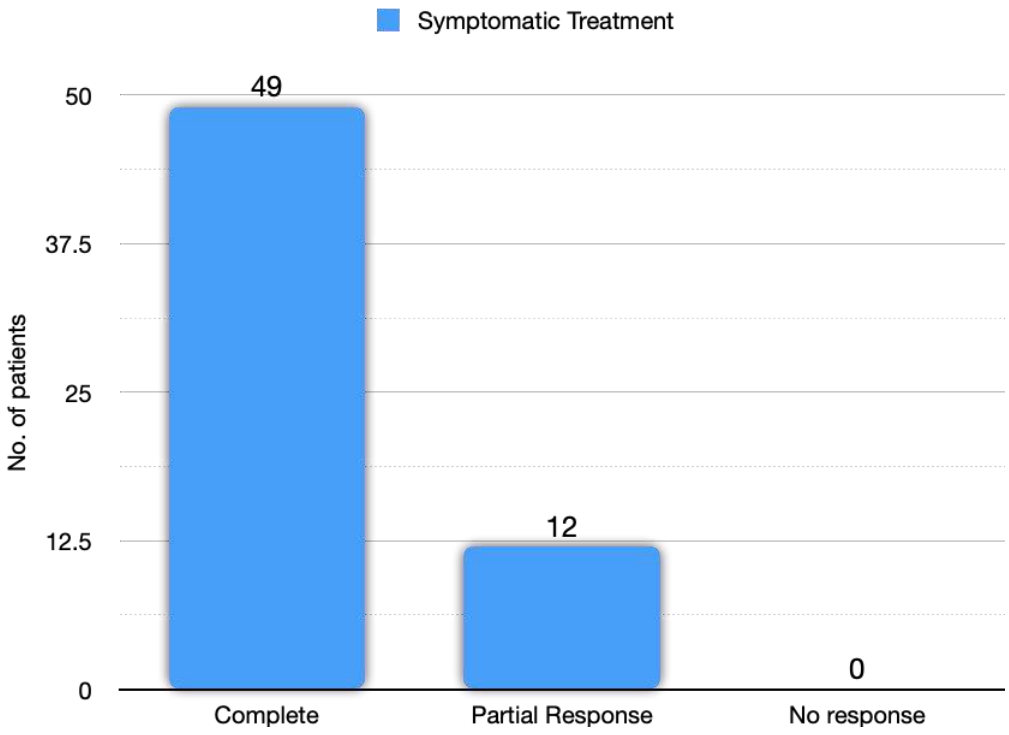

Figure 39: Bar graph showing distribution of response to various treatment modalities in patients with Thrombocytopenia due to Tropical Fever

Table 38: Distribution of response to various treatment modalities in patients with Thrombocytopenia due to Sepsis

	Steroid	Symptomatic Treatment	Steroid + TPO-RA + IVIG	Total
Complete	3	6	1	10 (52.63%)
Partial Response	0	4	0	4 (21.05%)
No response	3	2	0	5 (26.31%)
Total	6 (31.57%)	12 (63.15%)	1 (5.26%)	19
Lost to Follow up	-	-	-	4

Out of a total of 23 patients with Thrombocytopenia due to Sepsis, 4 were lost to follow up. Out of these 19 patients, 6 (31.57%) were given steroids. Half of these 6 patients showed complete response, whereas the other half showed no response.

12 patients (63.15%) were given only symptomatic treatment. Half of these patients showed complete response, whereas only 2 showed no response.

Only 1 patient was given a combination therapy of Steroid, TPO-RA and IVIG and that patient showed complete response.

10 patients with thrombocytopenia due to Sepsis showed complete response to treatment. Majority of patients (n=6, 60%) showed complete response to symptomatic treatment only. 30% of these 10 patients received steroids. Only 1 patient was given combination of all three treatment strategies (steroids + TPO-RA + IVIG)

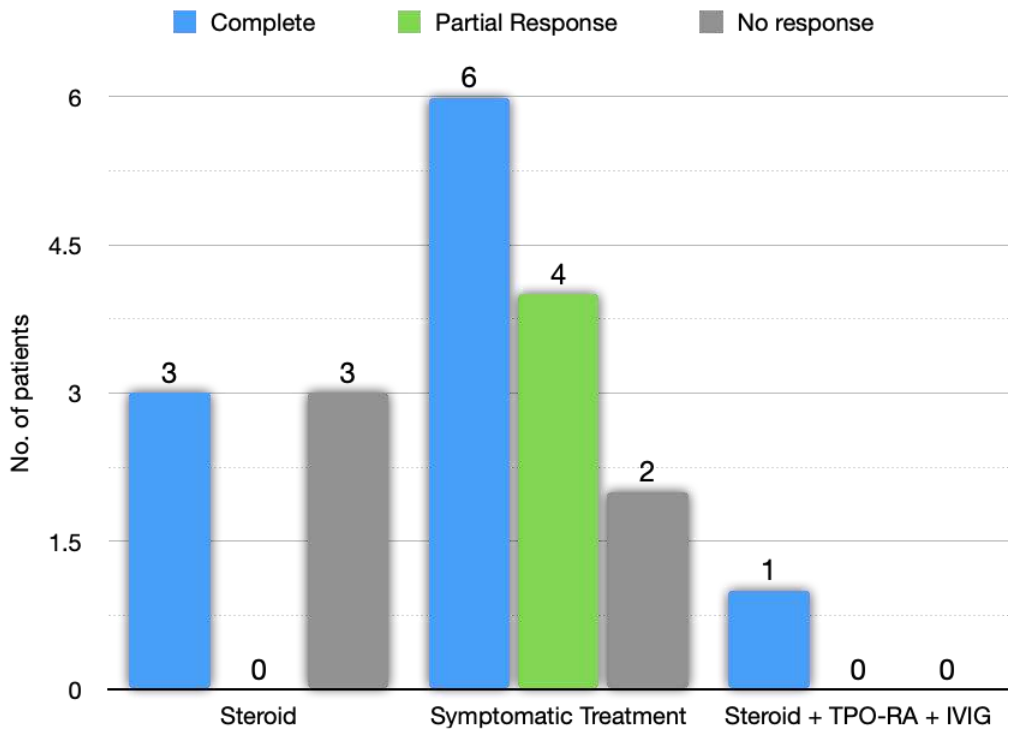

Figure 40: Bar graph showing distribution of patients given various treatment modalities according to the response

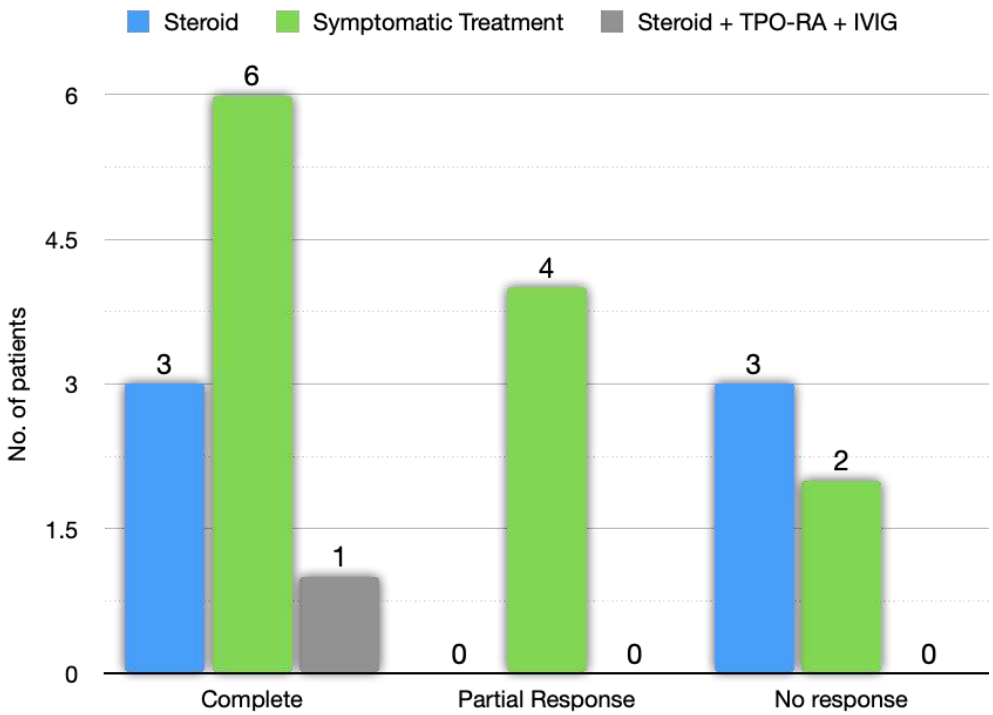

Figure 41: Bar graph showing distribution of patients with different response to treatment according to the treatment given

Table 39: Distribution of response to various treatment modalities in patients with Thrombocytopenia due to ITP

	Steroid	Steroid + TPO-RA	Steroid + TPO-RA + IVIG	Total
Complete	11	0	1	12 (66.67%)
Partial Response	4	0	0	4 (22.22%)
No response	1	1	0	2 (11.11%)
Total	16 (88.89%)	1 (6.25%)	1 (6.25%)	18

In these group of patients, majority of them (88.89%) received treatment with steroids only. 11 of these 16 (68.75%) showed complete response to this treatment and only one patient showed no response.

One of group required treatment with a combination of steroids and TPO-RA agents and showed no response.

One patient required combined treatment with steroids, TPO-RA agents and IVIG and showed complete response.

Among the 12 patients who showed complete response to treatment of Thrombocytopenia due to ITP, 11 were given steroids and 1 was given a combination therapy with steroids, TPO-RA agents and IVIG.

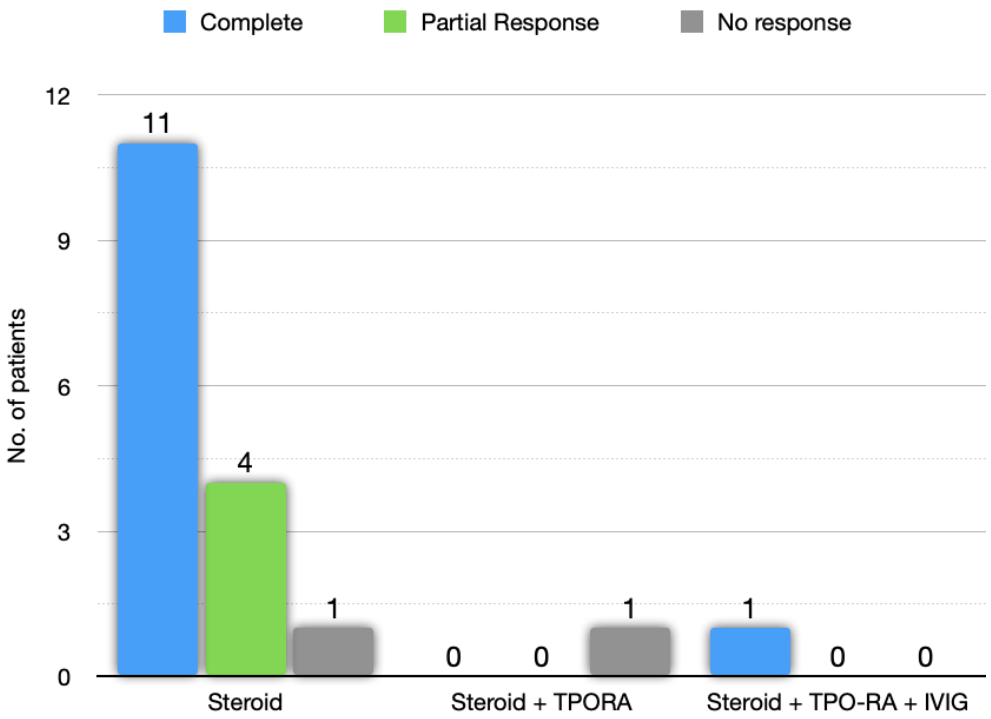

Figure 42: Bar graph showing distribution of patients with thrombocytopenia due to ITP and given various treatment modalities according to the response

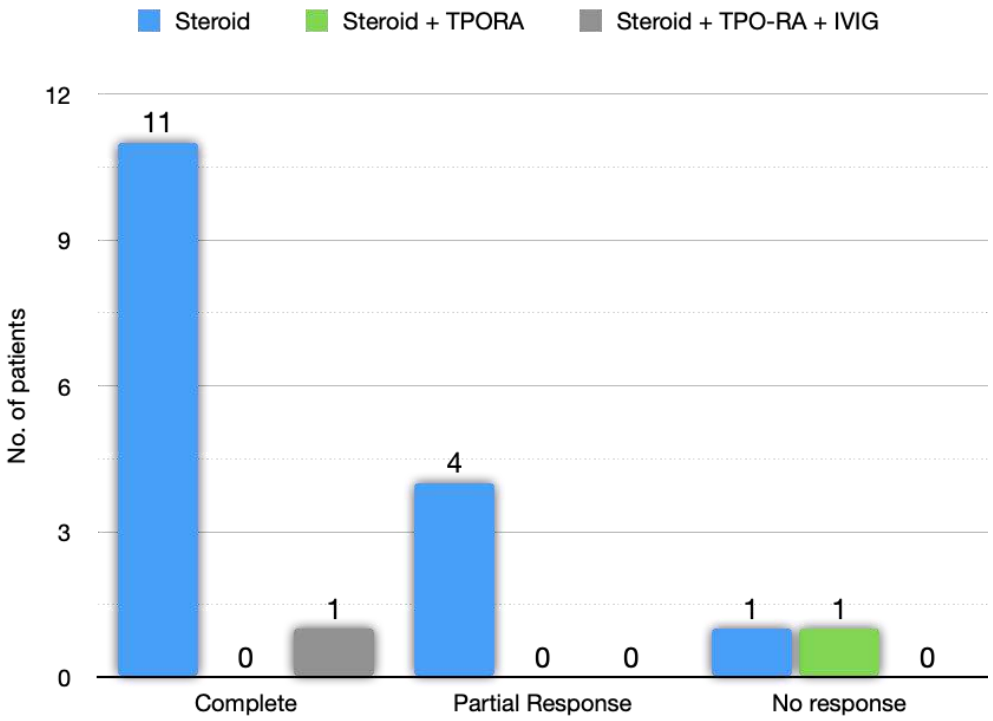

Figure 43: Bar graph showing distribution of patients with different response to treatment according to the treatment given

Table 40: Distribution of response to various treatment modalities in patients with Thrombocytopenia due to Autoimmune causes

	Steroid	Percentage
Complete	2	66.67%
Partial Response	1	33.33%
No response	0	0.00%
Total	3	

All patients with Autoimmune Thrombocytopenia were managed with Steroids. 2 patients out of 3 showed complete response whereas none of them showed no response to treatment.

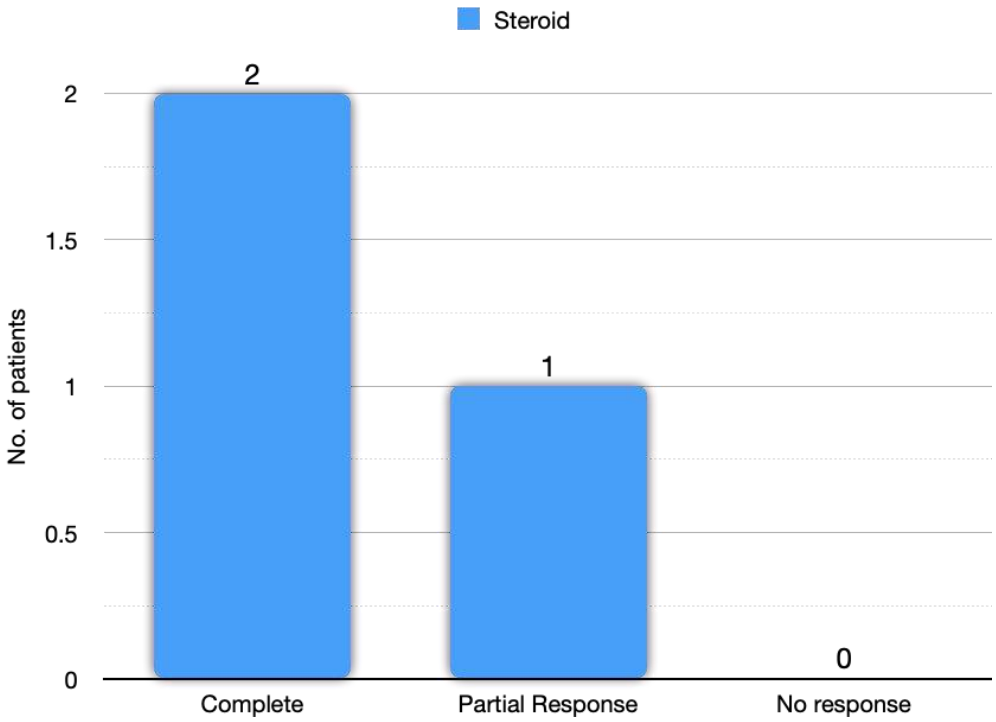

Figure 44: Bar graph showing distribution of patients with autoimmune thrombocytopenia according to response to the treatment given

Table 41: Distribution of response to various treatment modalities in patients with Drug induced Thrombocytopenia

	Symptomatic Treatment	Percentage
Complete	1	100.00%
Partial Response	0	0.00%
No response	0	0.00%
Total	1	

The one patient with drug induced Thrombocytopenia, showed complete response with symptomatic treatment only.

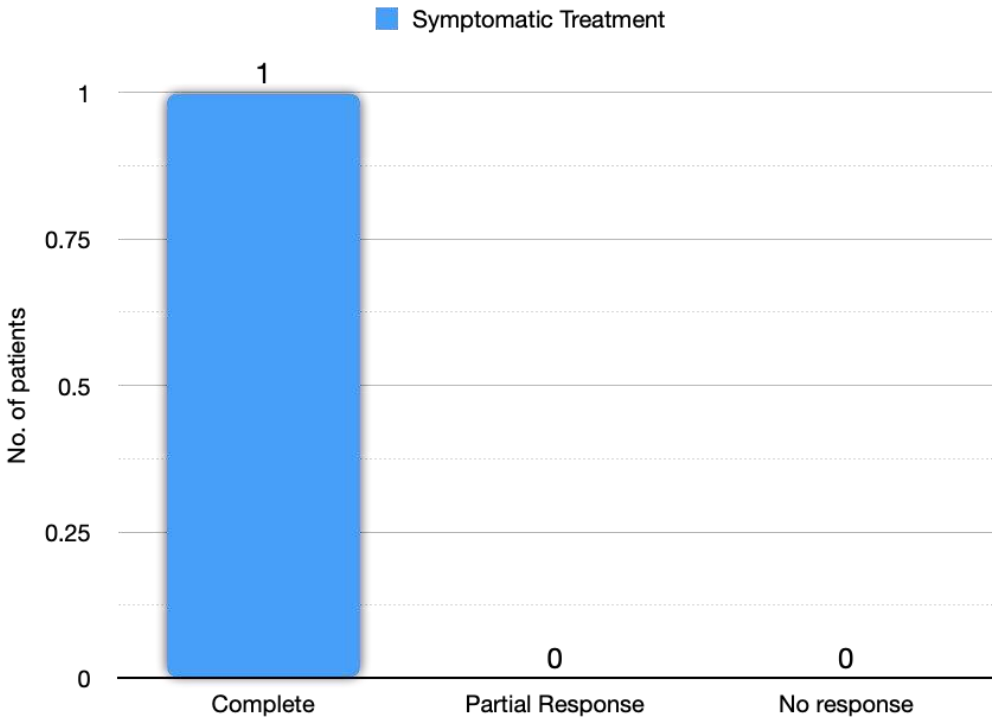

Figure 45: Bar graph showing distribution of patients with drug induced thrombocytopenia according to response to the treatment given

Table 42: Distribution of response to various treatment modalities in patients with Thrombocytopenia due to Tuberculosis

	Steroid	Symptomatic Treatment	Total
Complete	1	0	1 (50.00%)
Partial Response	0	0	0
No response	0	1	1 (50.00%)
Total	1 (50.00%)	1 (50.00%)	2
Lost to Follow up	-	-	1

One patient in this group was lost to follow up. Out of the rest of the 2 patients, one was given steroids and showed complete response. The other patient was managed with symptomatic treatment only and showed no response.

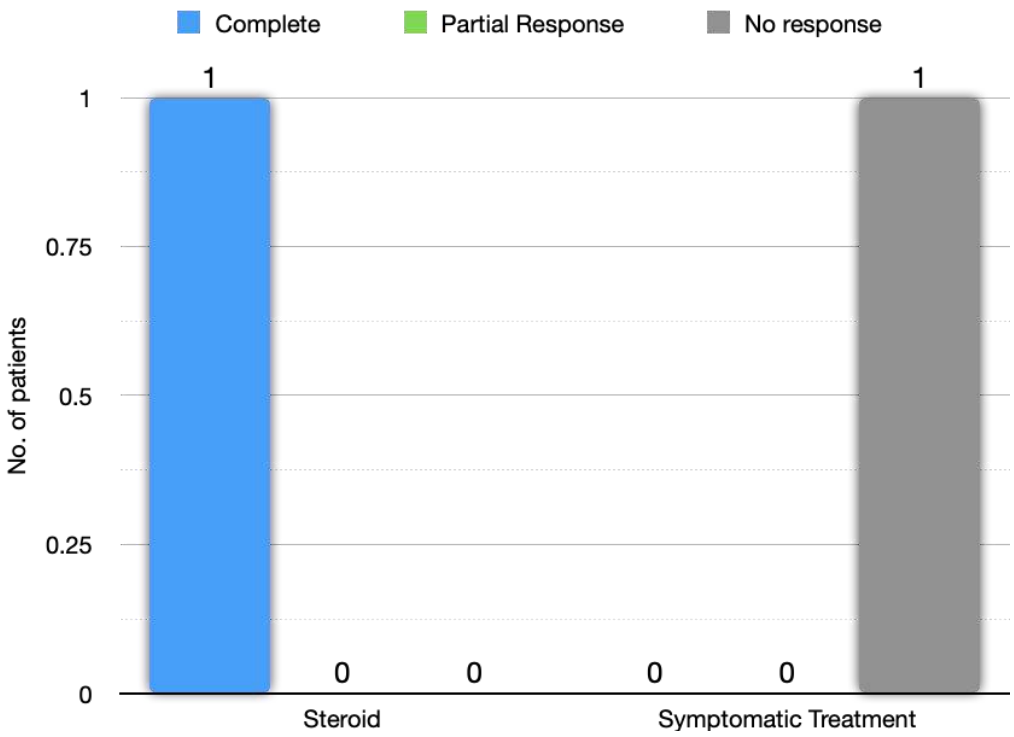

Figure 46: Bar graph showing distribution of patients with thrombocytopenia due to Tuberculosis and given various treatment modalities according to the response

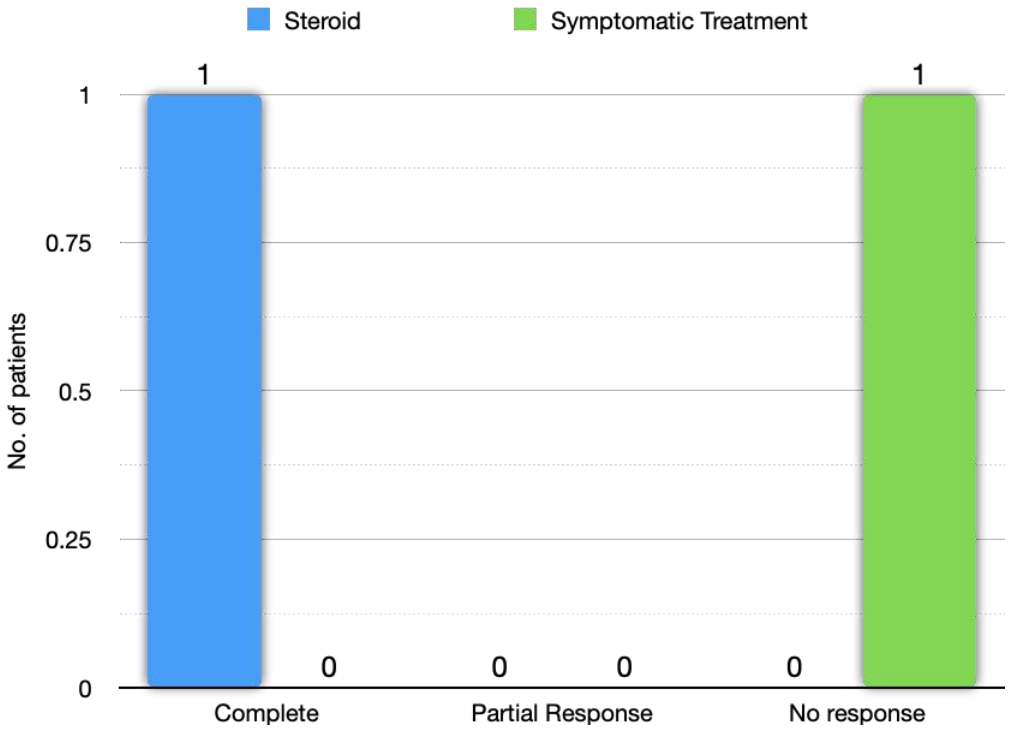

Figure 47: Bar graph showing distribution of patients with different response to treatment according to the treatment given

Table 43: Distribution of response to various treatment modalities in patients with Thrombocytopenia due to Megaloblastic Anaemia

	Symptomatic Treatment	Percentage
Complete	2	100.00%
Partial Response	0	0.00%
No response	0	0.00%
Total	2	

There were 2 patients with thrombocytopenia due to Megaloblastic Anaemia and both were given symptomatic treatment. Both patients showed complete response to treatment.

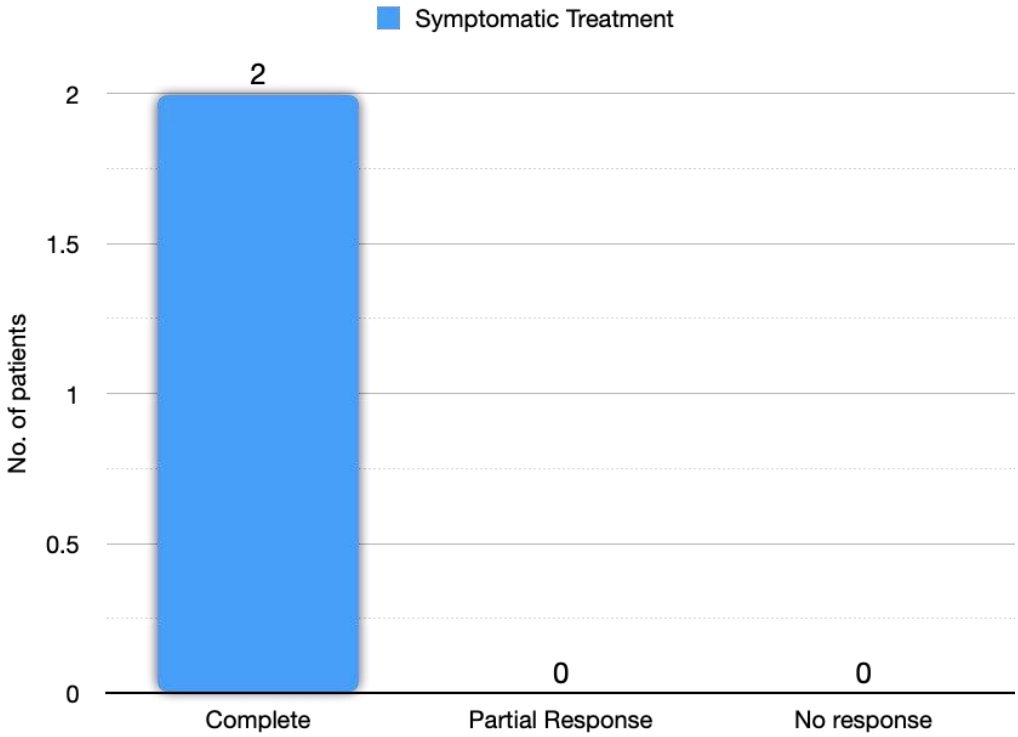

Figure 48: Bar graph showing distribution of patients with thrombocytopenia due to Megaloblastic Anaemia according to response to the treatment given

Table 44: Distribution of response to various treatment modalities in patients with Thrombocytopenia due to Multifactorial causes

	Steroid	Symptomatic Treatment	Steroid + IVIG	Total
Complete	3	0	0	3 (30.00%)
Partial Response	3	2	0	5 (50.00%)
No response	0	1	1	2 (20.00%)
Total	6 (60.00%)	3 (30.00%)	1 (10.00%)	10
Lost to Follow up	-	-	-	2

Two patients in this group were lost to follow up. 6 out of 10 remaining patients (60%) were treated with only Steroids and only half of them showed complete response to treatment.

3 out of 10 patients were managed with symptomatic treatment only and none of them showed complete response. 2 out of these 3 patients showed partial response to treatment and one showed no response.

One patient was given a combination treatment of steroids and IVIG and showed no response to treatment.

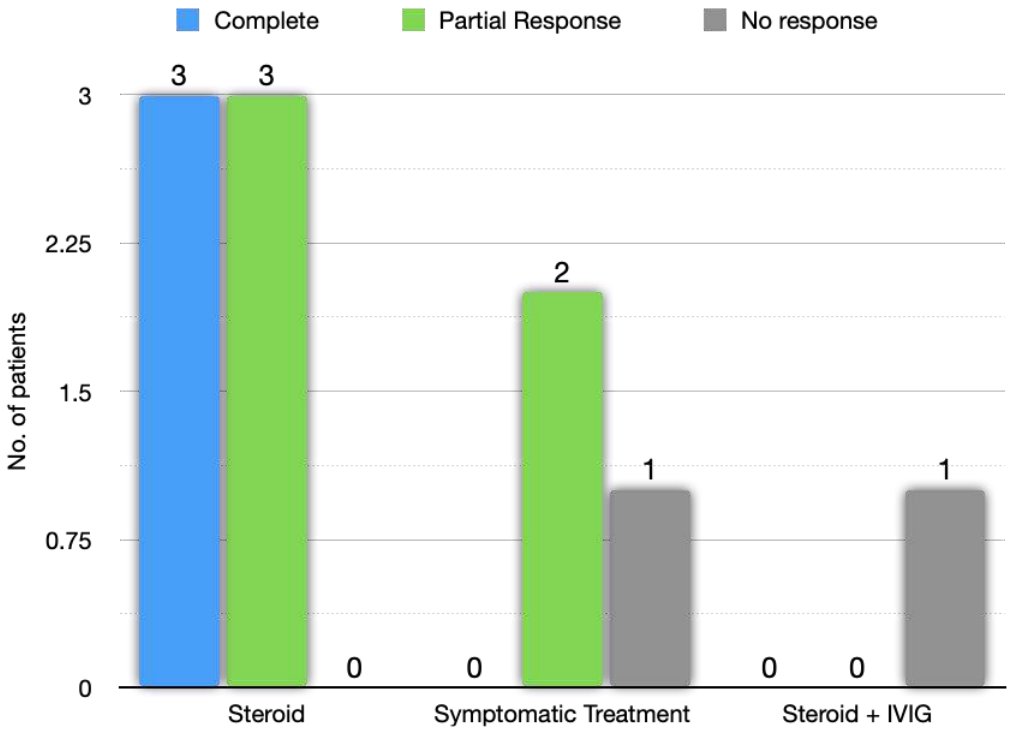

Figure 49: Bar graph showing distribution of patients with thrombocytopenia due to Multifactorial causes and given various treatment modalities according to the response

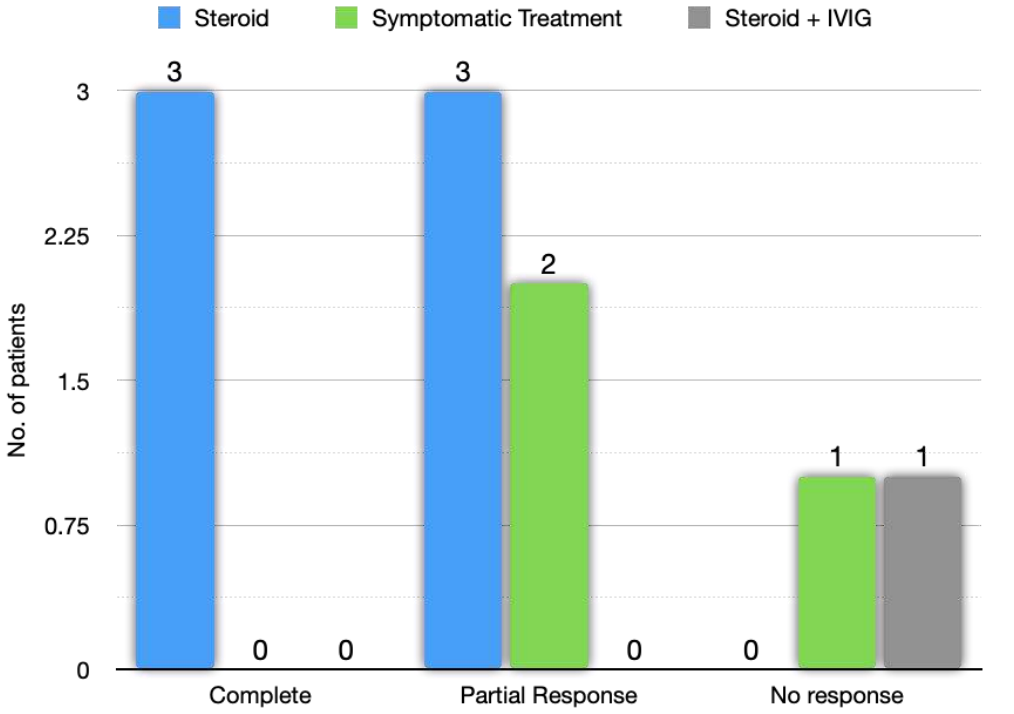

Figure 50: Bar graph showing distribution of patients with different response to treatment according to the treatment given

Table 45: Distribution of patients based on Steroids used in the study

Treatment modality used	No. of patients	Percentage
Dexamethasone	14	36.84%
Hydrocortisone	6	15.79%
Methylprednisolone	13	34.21%
Prednisolone	5	13.16%
Total	38	

Total 41 patients received steroids in our study. Dexamethasone and methylprednisolone were given to approximately one third each of the patients receiving steroids. 6 patients were given hydrocortisone. Hydrocortisone was not used as a part of pulse therapy it was given for the patients with refractory septic shock. 5 patients received Prednisolone in our study.

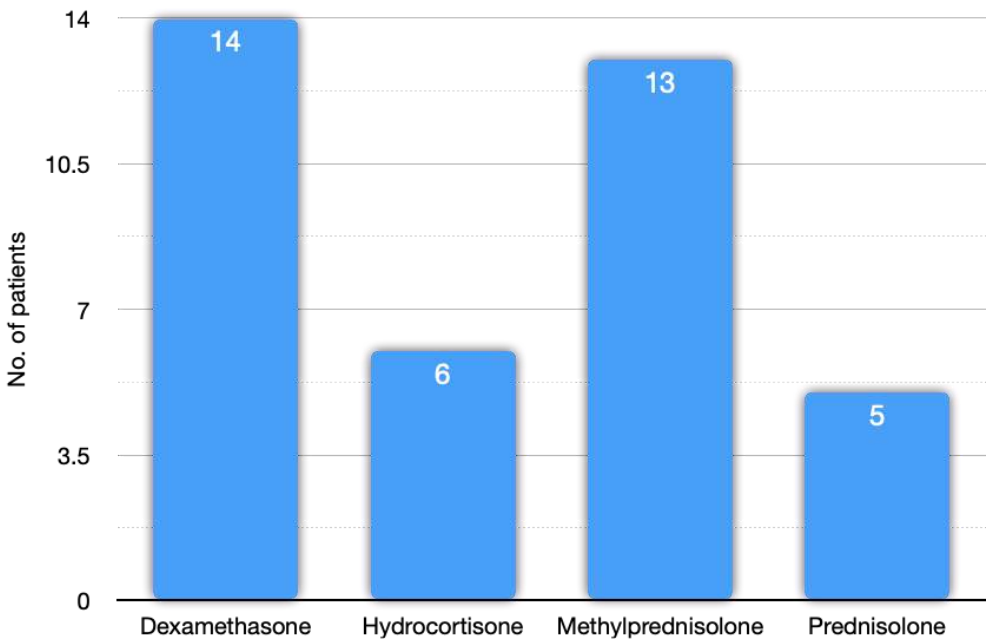

Figure 51: Bar graph showing distribution of patients based on Steroids used in the study

Table 46: Distribution of patients receiving TPO-RA agents

Drug used	No. of patients
Eltromobopag	1
Romiplostim	3

4 patients were given Thrombopoietin Receptor Agonists. 2 types of TPO RA were used which includes Eltromobopag and Romiplostim. 3 patients received romiplostim and only 1 patient received Eltromobopag in our study.

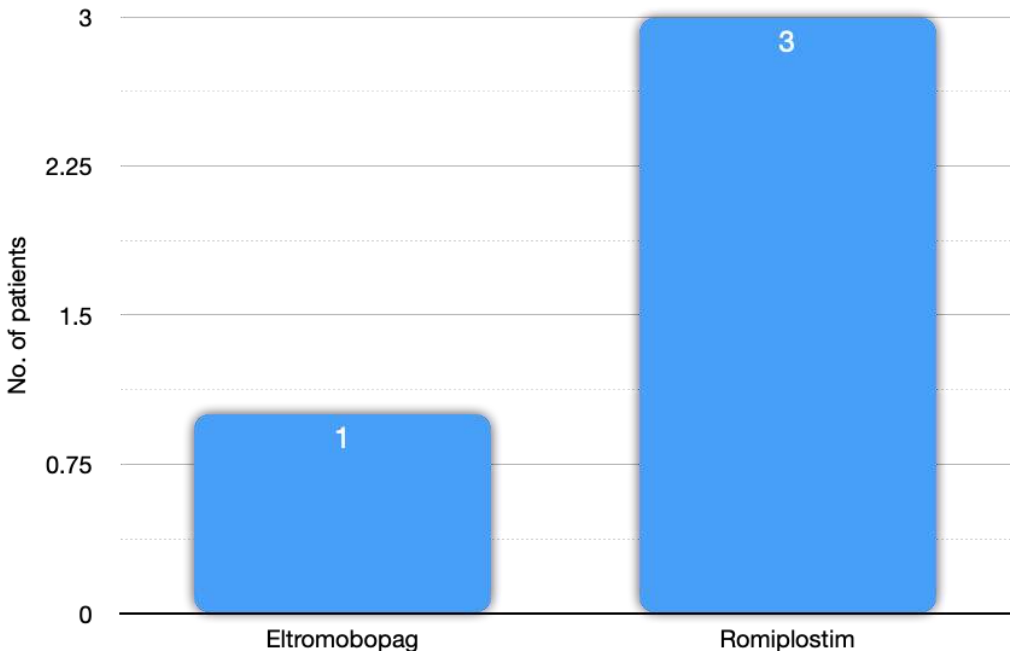

Figure 52: Bar graph showing distribution of patients receiving TPO-RA agents

Table 47: Distribution of patients according to response

Type of Response	No. of cases	Percentage
Complete	80	68.97%
Partial	26	22.41%
No response	10	8.62%

7 patients out of a total of 123 were lost to follow up and were not included in the analysis of response to various treatment modilities. Approximately two thirds of the patients available for follow up showed complete response.

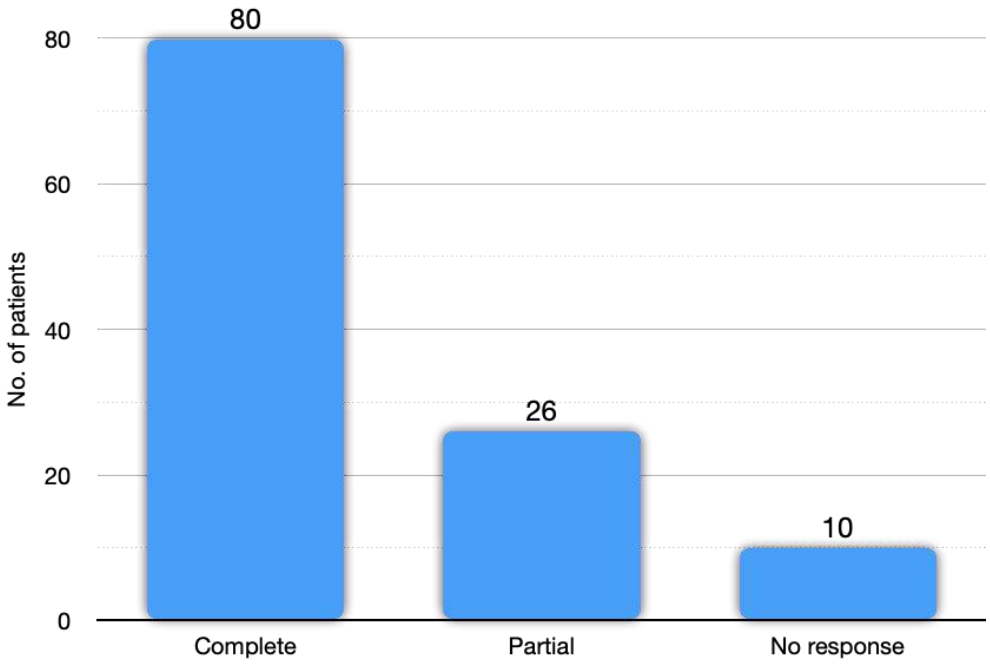

Figure 53: Bar graph showing distribution of patients according to response

Table 48: Mean number of days in which response was seen in various etiologies

Etiology	Mean no. of Days
ITP	4.59
Sepsis	5.5
Multifactorial	3.37
Tropical fever	3.18
Megaloblastic Anaemia	2.5
Tuberculosis	2
Drug induced	2
Autoimmune	7

Response (platelet counts of more than $30x10^9$/L) to treatment was shown earliest in patients with severe Thrombocytopenia due to Tuberculosis and Drug induced Thrombocytopenia. In Autoimmune Thrombocytopenia, response to treatment took an average of 7 days.

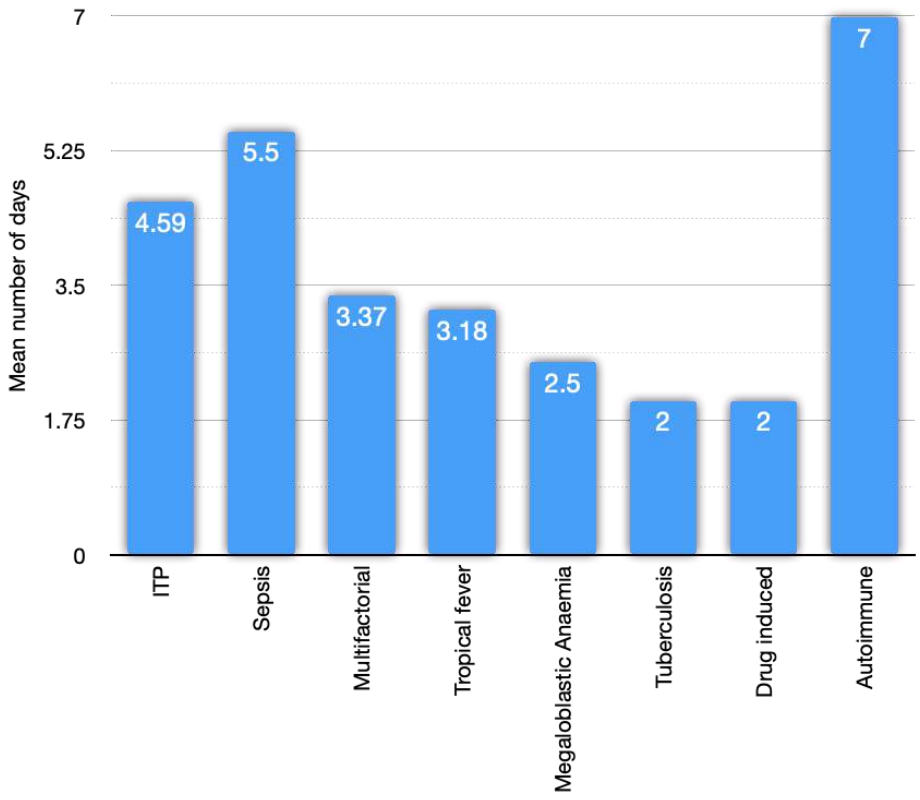

Figure 54: Bar graph showing mean number of days in which response was seen in various etiologies

Table 49: Distribution of patients with severe thrombocytopenia who needed ICU care

	No. of cases	Percentage
Yes	37	30.1%
No	86	69.9%
Total	123	100.0%

Out of a total of 123 patients, 86 (69.9%) did not require ICU care.

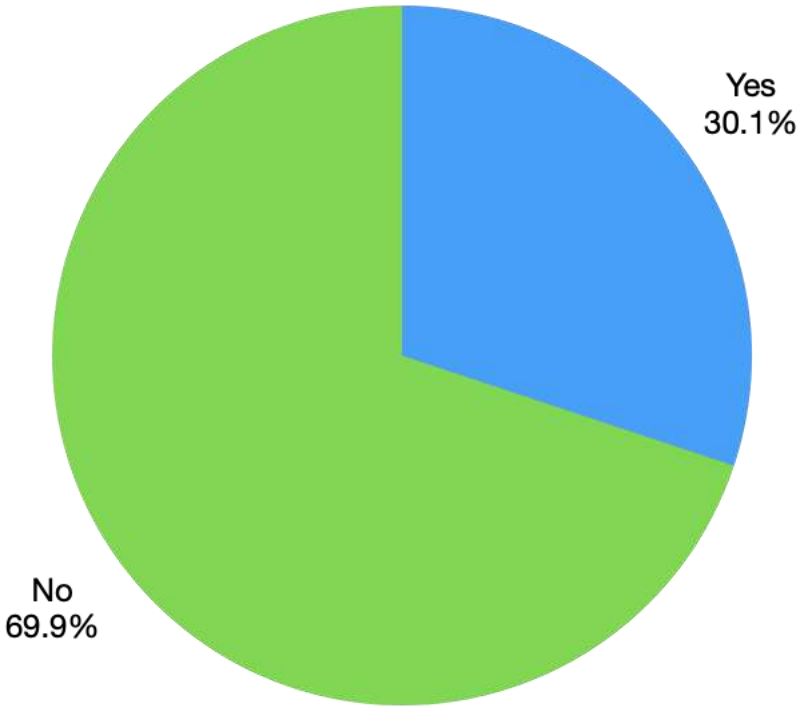

Figure 55: Pie chart showing distribution of patients with severe thrombocytopenia who needed ICU care

Table 50: Mortality distribution according to Etiology and response

Etiology	Complete	Partial	No Response
Sepsis	0	1	2
Multifactorial	0	0	1
Tropical fever	0	1	0

Among 116 patients, mortality was seen in only 5 patients (4.31%). Among these 5 patients, 3 patients (60%) were having Sepsis and only 1 of them showed partial response to treatment for thrombocytopenia. Mortality was seen in one patient each due to tropical fever (Dengue shock syndrome with ischemic Hepatitis) and multifactorial causes (Autoimmune cause in combination with respiratory sepsis). All patients with mortality were given extensive ICU care. Rest of the 111 were either discharged in stable condition (n=99, 85.34%) or discharged against medical advice for various reasons (n=12, 10.34%).

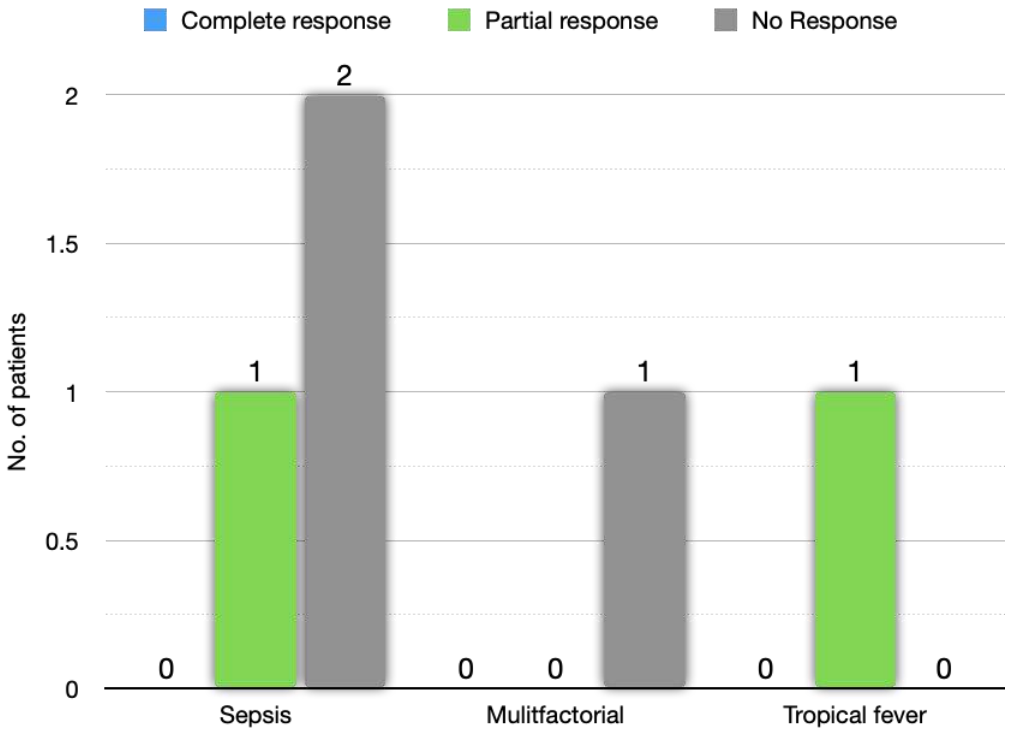

Figure 56: Bar graph showing distribution of patients with mortality with thrombocytopenia due to various etiologies according to the response to the treatment of thrombocytopenia

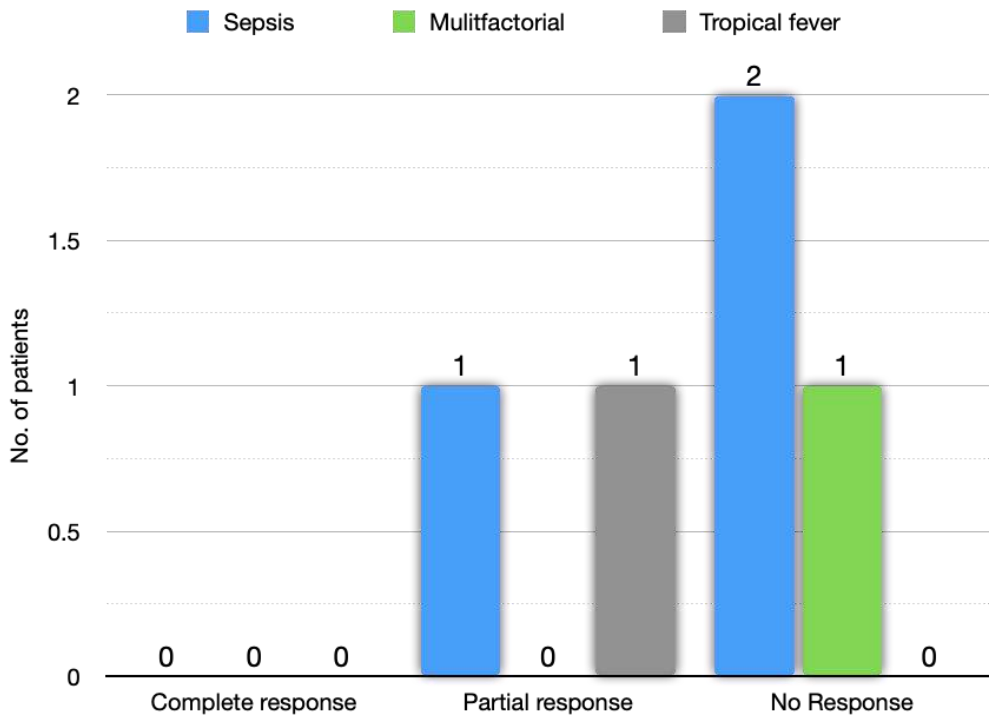

Figure 57: Bar graph showing distribution of patients with mortality with response to treatment of thrombocytopenia according to the etiology of thrombocytopenia

DISCUSSION

Diagnosis of Thrombocytopenia in itself is a challenge and management is primarily based on the cause of Thrombocytopenia. Severe thrombocytopenia (platelet count less than $20x10^9$/L) increases the morbidity and mortality of the patient. Improvement in thrombocytopenia is a good prognostic marker for the patients.

There is limited literature in the Indian context which has explored the causes and the clinical presentation of severe thrombocytopenia and its association with bleeding manifestations and mortalities.

In our study, a total of 123 patients with severe thrombocytopenia were enrolled. Approximately one fourth of the total patients in our study were in the age group of 19-30 years with a mean age 44.80 years. Majority of the patients were males (60.2%). Age and Sex distribution of other studies is as follows.

PICTURE 4: Age and sex distribution of different studies is as shown in the table:

Studies	Age Group	Mean age	Male:female
Our Study	19-30 years	44.80 years	1.3:1
Sridhar et al (35)	-	42.92 years	2.3:1
Lakshmikumar et al [16]	-	41.11 years	1.5:1
Gamit et al (36)	18-29 years	-	1.2:1
Mural et al (38)	18-30 years	-	1.5:1
Vimal et al [39]	41-60 years	-	1.4:1
Sale et al [41]	35-54 years	-	1.5:1

The variation in the age group seen in our study can be attributed to the diverse etiology associated with severe thrombocytopenia.

Most common etiology of severe thrombocytopenia in our study was Tropical fever which was seen in almost half of the cases, followed by Sepsis, seen in almost one-fourth of cases. The third most common cause was found to be Immune Thrombocytopenic Purpura (14.63%). There were 12 patients (9.76%) in whom severe thrombocytopenia was attributed to multiple etiologies.

Similar to our study, Sridhar et al [35] found Dengue fever as the most common cause of thrombocytopenia followed by Sepsis.

Gamit et al [36] also found Dengue as the most common cause of thrombocytopenia in their study.

In contrast, Lakshmikumar et al [16] concluded Septicemia followed by Dengue fever as the most common causes.

Septicemia was the most common cause of Thrombocytopenia in the studies done by Mural et al [38] and Zhang et al [40].

In half of our patients, Tropical fever was the only cause of Severe Thrombocytopenia. However, in 5 patients with Multifactorial Thrombocytopenia, Tropical fever was present in association with ITP, Autoimmunity, Sepsis and Hemophagocytosis. Most common infection in the Tropical fever group in our study was Dengue fever followed by Leptospirosis.

Choudhary et al [73] recorded Dengue fever followed by Malaria as the most common cause of febrile thrombocytopenia.

There were 5 patients in our study, observed to have dual Tropical fever infections. In this group, the patients had positive serology results for Dengue in association with Scrub Typhus, Malaria and Leptospira. One patient had positive serology for Enteric fever and Leptospira.

Dual infections were also seen in a study by Raina et al [74], who found associated Dengue fever and Scrub Typhus infections in almost two thirds of the patients. Other combination observed in their study were Leptospira with Scrub Typhus, Malaria with Scrub Typhus, Dengue fever with Scrub Typhus, Dengue fever with Chikungunya and Dengue fever with Scrub Typhus with Malaria.

Thus, we can conclude that Dengue fever is the leading cause of Severe Thrombocytopenia and can also present in association with other Tropical fever infections.

In our study, Sepsis was the primary cause of severe thrombocytopenia in 23 patients and was present as one of the causes in another 4 patients of the Multifactorial group. Sepsis occurred in association with Autoimmunity, Tropical fever and Hemophagocytosis in our study.

In studies done by Zhou et al [75] and Crowther et al [67] in patients with Sepsis managed in ICU setting, severe thrombocytopenia (<50x10^9/L) was seen in 19.7% patients and 2.3% patients respectively.

PICTURE 5: Incidence of severe thrombocytopenia in ICU patients observed in different studies.

	Zhou et al [75]	Crowther et al [67]
Total patients with Sepsis	249	261
Patients with Thrombocytopenia (<150)	90 (36.1%)	62(23.7%)
Moderate thrombocytopenia (50-100)	41(16.5%)	-
Severe thrombocytopenia (<50)	49(19.7)	6(2.3%)

Genitourinary sepsis was most common cause of severe Thrombocytopenia in our study followed by gastrointestinal sepsis. Respiratory sepsis was seen as a cause of severe Thrombocytopenia in 3 patients in our study. One patient presented with Sepsis induced severe Thrombocytopenia due to Infective Endocarditis.

Zhou et al [75] concluded that severe Thrombocytopenia was associated more often with intra-abdominal or blood stream infection than with the respiratory infection.

Major source of sepsis causing thrombocytopenia in the study by Sharma et al [76] included pneumonia (26 patients), urinary tract infection (16 patients), skin/decubitus infections (9 patients), intra-abdominal infections (5 patients), central nervous system infection (three patients) and infection from unknown sources (10 patients).

Most of the patients with sepsis induced thrombocytopenia in study by Sridhar et al [35] were diagnosed with pneumonia as the primary cause of sepsis (46.5%).

In another study by Venkata et al [77], pneumonia was seen in 38.8% of patients as the cause of thrombocytopenia.

In conclusion, Sepsis is one of the leading causes of severe Thrombocytopenia. However, there is no consensus on the source of Sepsis which can frequently cause severe Thrombocytopenia. In our study Respiratory infections were not a common cause of Thrombocytopenia as observed by most other studies.

There were 18 patients (14.63%) with severe thrombocytopenia diagnosed with primary Immune Thrombocytopenic Purpura. Mean age of patients with ITP in our study was 44.17 years with 11 males and 7 females.

Wong et al [78] studied 78 ITP patients, majority of them were females and belong to the age group of 20-39 years.

Spahr et al [79] reviewed 40 patients with male to female ratio of 1:3 with the majority (42%) of patients in 20-40 years age group.

The mean initial platelet count was 15 x 10^9/L in women and 8 x 10^9/L in men (p= 0.03) in a study by Frederiksen et al [80].

There were 6 patients in multifactorial etiologies who were diagnosed with secondary ITP. ITP in multifactorial etiology was seen secondary to Tropical fever, Autoimmune etiology and both.

In a study by Palau et al [81], secondary ITP was diagnosed in 51 of the total 484 ITP patients, most commonly associated with SLE (n=9, 17.6%) and Lymphoproliferative disorders. Infections constituted the third most common cause of Secondary ITP in their study.

In our study, bone marrow examination was done in all patients of ITP which suggested hyper destructive thrombocytopenia.

In a retrospective analysis done by Mak et al [53], bone marrow examination of 83 patients was suggestive of active marrow due to peripheral destruction. 81 of them were diagnosed with primary ITP and the remaining 2 were diagnosed with SLE.

There were 7 patients in our study with Autoimmune component as a cause of Thrombocytopenia. Almost half of them (n=3) had autoimmunity as the only cause of severe Thrombocytopenia whereas in rest of the patients, autoimmunity was associated with other

etiologies like Sepsis, Tropical fever and Hemophagocytosis as mentioned above in Table 7.

ANA profile was positive in all of them. More than half of the patients (57%) were positive for SSA/Ro 52 antibody. Other antibodies seen in our study included DsDna, Lupus anticoagulant and ku/uctd. Bone marrow was done in 6 patients and was suggestive of hyper destructive marrow.

In a study done by Costa Pires et al [82], 707 patients of SLE were followed up to 40 years. Less than one fourth of the patient (n=162, 22.9%) was having thrombocytopenia prior to or during the course of SLE. 14.2 %(n=23) patients had isolated thrombocytopenia before the diagnosis of SLE. More than half the patients (n=98, 60.5%) developed asymptomatic thrombocytopenia and only 6 patients (3.7%) had major bleeding events. They concluded that development of severe thrombocytopenia in SLE was associated with an increased risk of death.

In a retrospective study done by Jung et al [83] in 230 SLE patients with thrombocytopenia, patients were divided into three groups based on mild (>50,000/mm^3), moderate (>20,000/mm^3 to <50,000/mm^3) and severe thrombocytopenia (<20,000/mm^3). Thrombocytopenia developed in a mean duration of 23.7±58.1 months after SLE diagnosis.

In our study one patient was found to have Drug induced severe thrombocytopenia with Methotrexate. Patient had previous history of Rheumatoid Arthritis for which she was taking Methotrexate for 2 months prior to presentation to our hospital.

In a study done by Lalevee et al [84], 433 cases of cytopenia associated with methotrexate were analyzed. They found that, 19.4% were due to medication errors, 39% patient were exposed to only methotrexate and 41.6% patients were exposed to methotrexate and other drugs.

There are many other drugs which can cause thrombocytopenia as has been mentioned in a study by George et al [85]. They analyzed cases of Drug induced thrombocytopenia and also devised a set of four clinical criteria to assess the likelihood of Drug induced thrombocytopenia.

About 51 drugs are considered “definite” and 17 others as “probable” causes of Drug induced thrombocytopenia. Classes of drugs causing thrombocytopenia include the Cinchona alkaloids like Quinine and Quinidine, NSAIDs, various antibiotics, especially Sulfamethoxazole and Vancomycin, anticonvulsants and sedatives, and the platelet inhibitors Tirofiban, Eptifibatide and Abciximab.

There were 3 patients with severe thrombocytopenia due to Tuberculosis. The diagnosis of Tuberculosis in two patients was made on basis of Bone marrow findings suggestive of granulomatous inflammation.

In a retrospective study done by Hakawi et al [86], out of 394 patients diagnosed with extra pulmonary tuberculosis, 22 patients (5.9%) were identified as having tuberculosis of the bone marrow. Thrombocytopenia was seen in 45% of the patients.

Two patients had severe thrombocytopenia due to Megaloblastic Anaemia. One of them had pancytopenia and the other one had bicytopenia.

In a study by Huang et al [87], 1553 patients with Megaloblastic anaemia were studied, thrombocytopenia was seen in 12% of the cases and pancytopenia was seen in 39.3%.

In our study, only 12 patients (9.8%) were found to have a positive Alcohol history. Out of these, three fourths of the patients presented with Sepsis induced severe Thrombocytopenia.

In the study done by Latvala et al [52] thrombocytopenia and combined cytopenia were a common finding in alcoholic patient. The severity of thrombocytopenia associated in alcoholic patients was not mentioned in the study.

Severe thrombocytopenia in our study was associated comorbid conditions in 65% of the patients. Type II Diabetes Mellitus (30 patients) and Hypertension (24 patients) were the commonest. Others include Thyroid disease, CAD, CVA, CKD, CLD and Rheumatoid Arthritis.

The most common symptom seen in the patient with severe thrombocytopenia in our study was fever which was seen in three

fourths of the total patients. Malaise/fatigue/generalized body weakness was seen in almost one third of the total patients.

Similarly, in the studies by Lakshmikumar et al [16] and Vimal et al [39], fever was the predominant symptom seen in all the patients with thrombocytopenia

Most common clinical feature seen in patients with Tropical fever in our study was fever followed by generalized malaise.

Khan et al [88] and Choudhary et al [73] found most common symptom in Dengue was fever followed by malaise.

Bleeding manifestations were seen in less than half (43.08%) of the patients. Ecchymosis was seen in maximum number of patients (19.51%) followed by Malena (17.07%), Gum bleed (17.07 %), Menorrhagia (6.50%) and Hematuria (5.69%). No incidence of any major bleeding event was observed in any patient in our study.

In the study of Lakshmikumar et al [16], 43.7% of total patients had bleeding events. Most common event was venipuncture bleed followed by gum bleed.

In the study of Sridhar et al [35], 23% of the cases had some sort of bleeding event. Malena was the most common bleeding manifestation observed.

In the study of Mural et al [38], 19% of the total patients showed bleeding events, most common bleeding manifestations was seen in Skin and Mucous membrane followed by Gum bleed and Malena.

In the studies of Vimal et al [39] and Yadav et al [42], bleeding manifestations were seen in 13.3% and 16.27% patients respectively.

In our study, one fourth of the patients with severe thrombocytopenia due to Tropical Fever presented with bleeding manifestations and the most common bleeding manifestation was Malena followed by Gum bleeding.

In the study of Ahmed et al [89], most common bleeding manifestation in patients with thrombocytopenia due to Dengue fever was Gum bleeding (67.9%) followed by Malena (51.5%).

Bleeding events were seen in 73.0% of Dengue patients in a study done by Lee et al [90].

In the Immune Thrombocytopenic Purpura group, three fourths of the patients in our study had bleeding manifestations. Most common was Gum bleeding (38.89%) followed by Ecchymosis (27.78%).

In the study by Hato et al [91], most common bleeding manifestation in ITP was Purpura (64.80%) followed by Oral mucosal bleed (20.27%).

In contrast, in the study of Wong et al [78], out of a total 78 patients, almost 22% (n=17) of the patients were asymptomatic. However, on presentation more than half (n=45) presented with petechiae / gum bleeding / epistaxis, followed by Menorrhagia (9 patients) and Hematuria (2 patients). 5 patients presented with major hemorrhagic events – four with gastrointestinal hemorrhage and one with intracranial hemorrhage.

In thrombocytopenia due to Sepsis, less than half (43.47%) of the patients presented with bleeding manifestation and most common bleeding manifestation was Ecchymosis (26.09%).

Among patients with Autoimmune etiology, 6 out of the 7 patients had bleeding manifestation. Most common bleeding manifestation seen in patients with autoimmune etiology includes Ecchymosis followed by Malena.

In the study of Jung et al [83] in 230 SLE patients with thrombocytopenia, there was no significant difference in clinical or laboratory findings among the groups according to severity of thrombocytopenia. However, haemorrhagic complications were more frequent in severe thrombocytopenia ($P < 0.001$).

In patients with severe thrombocytopenia due to Tuberculosis and Megaloblastic Anaemia, no bleeding manifestation were observed.

However, in patient with Drug induced Thrombocytopenia due to Methotrexate, patient presented with oral ulcers and Malena. On laboratory evaluation, pancytopenia was also seen.

Management of patients with severe thrombocytopenia depends on the underlying disease. Different treatment modalities used in our study were symptomatic management including transfusion of RDP and/or SDPs and various drugs like Steroids, Thrombopoietin Receptor Agonists (TPO-RA) and IVIG.

Almost 80% of the total patients (n=100) in our study received Platelet Transfusions in the form of SDP, RDP or both as per the recommendation that platelets can be transfused prophylactically in patients with severe thrombocytopenia.[56]

In study of Sridhar et al [35], one fourth of the total patients (n=26) received platelet transfusion, out of which 23 patients were given transfusion therapeutically and 3 were given prophylactically.

Almost one third of the total patients (n=38) received Steroids in our study. Thrombopoietin Receptor Agonists (TPO-RA) were received by 4 patients and IVIG by 3 patients.

Patients with severe thrombocytopenia due to Tropical Fever were managed symptomatically. Almost 90% of the patients received platelet transfusions. None of the patients received any drugs for thrombocytopenia including steroids, IVIG or TPO-RA in our study.

After management with symptomatic treatment including transfusions, complete response was seen in 49 patients out of 61 (80.32%) and partial response was seen in 12 patients (19.67%). There was no patient with severe thrombocytopenia due to tropical fever showing no response.

In a study done by Furtado et al [92], platelet transfusions were given prophylactically to all the patients with severe thrombocytopenia due to Dengue fever (n=6). None of the patient in their study received steroid or IVIG.

In conclusion, thrombocytopenia due to Tropical fever is usually self-limiting and platelet counts can be improved with transfusions, if required.

In patients with severe thrombocytopenia due to Sepsis, we found that the management of sepsis including the use of appropriate antibiotics and removing the source of the infection plays a major role in the treatment of thrombocytopenia.

Out of the total of 23 patients, four patients were lost to follow up, thus response was analyzed in the remaining 19 patients. More than 80% patients with sepsis received platelet transfusions in our study.

Steroids were given in only 6 patients, half of them showed complete response and the other half showed no response.

There were 12 patients in whom none of the thrombocytopenia specific drugs including steroids, IVIG or TPO-RA were given, among them half of the patients showed complete response, one third had partial response and only 2 patients had no response.

There was one patient with persistent thrombocytopenia despite adequate treatment of Sepsis. Bone marrow examination showed normocellular marrow with erythroid prominence. Due to persistent thrombocytopenia, the patient was given a combination therapy with Steroids, Romiplostim and IVIG and complete response was achieved. After 20 days of hospital stay, he was discharged in stable condition.

In a study done by Venkata et al [77], the 28-day mortality was similar between patients with and without thrombocytopenia. Mortality was seen in 17.4% of the patients with resolved thrombocytopenia and 54.2% patient with no resolution of thrombocytopenia. They concluded that non resolution of thrombocytopenia, not thrombocytopenia itself was associated with increased 28–day mortality.

In a study of 72 patients with severe sepsis by Qin Wu et al [93], 38 patients received TPO-RA and 34 patients were kept in the control group. Faster improvement of platelets was observed in the TPO-RA group with a significant difference.

Thus, we can conclude that the management of the underlying sepsis is the key strategy in Sepsis induced thrombocytopenia and other treatment modalities can be used if thrombocytopenia persists.

Steroids were the main treatment modality in ITP patients and almost 60% of the patients were given platelet transfusions. There were many formulations that were used including oral and intravenous Methylprednisolone (alone or in combination). Mean duration of pulse therapy given was 3 days for both Methylprednisolone and Dexamethasone. Patients, after intravenous steroid therapy, were shifted to oral steroids (Prednisolone) which was tapered off after two weeks. Steroids were given in all the patients of ITP wither alone or in combination with other agents, in our study. There were 16 patients

with ITP in whom steroids were given alone, more than two thirds of them showed complete response (68.75%), less than one third showed partial response (25%) and only one patient had no response (6.25%).

Chang et al [64] studied the response of steroids in ITP patients and complete response was seen in two third patients (65.2%), partial response in less than one third (23.3%) and no response was seen in 11.4%.

Other studies also report the rate of response in the range of 70-80 %. [94, 95,96]

In our study, high dose Methylprednisolone was used initially followed by maintenance with conventional dose Prednisolone in one third of the total patients with ITP (n=18). All these patient achieved complete response and were discharged in stable condition.

In a study by Alpdogan et al [95], it was observed that high dose Methylprednisolone (30 mg/kg/day) is superior to conventional doses of Prednisolone as first line treatment.

Dexamethasone was used alone as an intravenous formulation in 8 patients for a mean of 3 days. With this therapy, complete response was seen in 87% of the patients (n=5), partial response in 2 patients and no response in 1 patient.

Chang et al [64] found that with a single course of Dexamethasone, used in 125 patients of ITP, the response rate was 85%.

In study by Depré et al [97], forty-four patients (11%) were treated with Dexamethasone and 60% patients responded while 40% showed no response.

In our study conventional dose of corticosteroid (1-2mg/kg/day) was given in 3 patients and low dose corticosteroid (0.25mg/kg/day) was given in 1 patient. All three patients with conventional dose of corticosteroid showed response while no response was seen with low dose corticosteroid.

In a study by Depré et al [97], a total of 92 patients (23%) received Prednisolone for ITP for at least one month. Only three fourth of patients responded to this treatment and no response was seen in one fourth of the patients.

Bellucci et al [98] compared conventional doses of corticosteroids with low doses for 3 weeks duration and found no difference in the rate of response between the two therapies.

In the study by Mashhadi et al [96], they concluded that conventional corticosteroid therapy is superior to high dose Dexamethasone.

One patient received a combination therapy with Steroid and TPO-RA and no response was achieved.

A combination of Steroids, and IVIG together were given in one patient and complete response was achieved.

TPO-RA were given in 4 patients in our study (2 patients with ITP, 1 patient with Sepsis and one patient with Tuberculosis). Eltromobopag was given in one patient with ITP in whom no response was seen. Romiplostim was given along with IVIG in one patient with ITP and one with Sepsis. Both patients achieved complete response.

Romiplostim was also given to a patient with Tuberculosis but the patient was lost to follow up.

Almaguar et al [99] used Eltromobopag in combination with oral Dexamethasone as frontline therapy in 12 patients in an outpatient setting. They achieved complete response rate in 83.3% and partial response rate in 16.7%.

In another study done by Haselboeck et al [100], treatment with Eltromobopag increased platelet count in 10 out of 11 patients.

Cooper et al [101] did an indirect comparison of two trials of Romiplostim and Eltromobopag using Bayesian meta regression, and suggested that Romiplostim significantly improves overall platelet response rates compared to Eltrombopag, but the results were not significant.

In the study by Depré et al [97], three fourth patients responded (n=35) with Romiplostim and one fourth showed no response (n=10). Almost 71% of the patients (n=45) responded with Eltromobopag and 29% showed no response (n=18). Three fourth patients responded (n=133) with IVIG and one fourth showed no response (n=32).

In our study, IVIG was used in combination with steroids and TPO-RA in two patients (one with ITP and the other with sepsis) and achieved complete response.

In a study by Spahr et al [79], it was concluded that IVIG is safe agent for increasing platelet counts rapidly as well as for controlling symptoms.

Apte et al [102] studied safety and efficacy of IVIG, and found that IVIG achieved a response rate of 76.5% in ITP patients.

In the study by Depré et al [97], three fourth patients responded (n=133) with IVIG and one fourth showed no response (n=32).

Steroids were given in all the 3 patients with severe thrombocytopenia due to Autoimmune causes, and response was seen in all the patients. Two thirds of the patients showed complete response and one third showed partial response. All of the patients in this group received platelet transfusions.

In a study done in SLE patients with thrombocytopenia by Nossent et al [103], all the patients who were given high dose of corticosteroids responded except one.

In a study by Arnal et al [104], response to treatment was assessed retrospectively in 59 patients with SLE associated thrombocytopenia. Complete response with steroid was seen in 28 patients and partial response was seen in 12 patients. High dose Methylprednisolone resulted in complete response in 4 patients and partial response in 2 patients.

In conclusion, thrombocytopenia due to ITP and Autoimmune causes can be effectively managed with high dose pulse therapy of steroids followed by maintenance doses. TPO-RA and IVIG can be used as adjuncts to steroid therapy.

There were three patients with thrombocytopenia due to Tuberculosis. Two third patients received platelet transfusion. One patient was lost to follow up and was not included in the outcome analysis. One patient was given steroid along with Antitubercular Treatment and showed complete response. The second one was managed with only Antitubercular Therapy and no response to thrombocytopenia was seen.

Al-Majed et al [105] studied 846 cases with active tuberculosis, out of which 9 patients (1%) had thrombocytopenia between 4-21x10^9/L.

They observed that the bone marrow examinations of these patients showed increased megakaryocytes. The platelet counts became normal after 2-6 weeks of administration of oral Prednisone combined with anti-tuberculous drugs

In study by Hakawai et al [86], adjunctive corticosteroid therapy was used in 4 patients along with anti-tuberculosis drugs.

Thus, we can conclude that steroids are a useful adjunctive therapy with Antitubercular Therapy for treatment of Tuberculosis induced thrombocytopenia

No specific treatment for decreased platelets was given in Drug induced Thrombocytopenia and complete response was achieved by stopping Methotrexate and was managed with Folinic acid. As mentioned earlier, treatment of this group of thrombocytopenia is recognition and stoppage of the offending agent. Transfusions can be given for quicker recovery of platelet counts in patients with increased risk of bleeding events.

Severe thrombocytopenia due to Megaloblastic Anaemia was seen in 2 patients. Both the patients received blood transfusions and showed complete response after managing the underlying condition.

In a cross-sectional study of etiological spectrum of pancytopenia by Santra et al [106], Megaloblastic anaemia was found to be treatable and reversible cause.

Out of 116 patients that were available for outcome analysis, mortality was seen in only 5 patients (4.31%). Among these 5 patients, 3 patients (60%) had Sepsis induced thrombocytopenia.

Mortality rate in the studies of Yadav et al [42] and Furtado et al [92] were 19.84% and 2.7% respectively.

Mortality was seen in one patient of Tropical fever group due to Dengue shock syndrome with ischemic Hepatitis, and one of Multifactorial group due to respiratory sepsis. All patients with mortality were given extensive ICU care.

Thus, mortality was maximum in Sepsis induced thrombocytopenia similar to the findings in the studies of Sridhar et al [35] and Yadav et al [42].

In conclusion, the mortality in Thrombocytopenia is usually Sepsis related and mortality due to other causes of thrombocytopenia are uncommon if severe bleeding manifestations can be prevented successfully.

SUMMARY

1. Majority of patients in our study were males with male: female ratio of 1.3.1 belonging to the age group 19-30 years.
2. Tropical fever (49.5%) was found to be the leading cause of thrombocytopenia followed by sepsis (18.7%) and ITP (14.63%).
3. Fever was the predominant presenting complaint (73.25%) followed by generalized body weakness (22.8%).
4. Bleeding manifestations were seen in less than half (43.08%) of the total patients.
5. Bone marrow examination was done in one third of the patients with severe thrombocytopenia. Two third of them showed hyperdestructive thrombocytopenia.
6. Complete response was seen in 80 % of the Tropical fever with symptomatic management.
7. Approximately half of the patients (52.63%) with sepsis showed complete response. 60% of the patients with complete response were given symptomatic management and I/V Antibiotics.
8. All the patients with ITP induced severe thrombocytopenia were given steroids. Two third of the total patients showed complete response.
9. Steroids were given in both the patients with severe thrombocytopenia due to autoimmune cause leading to complete response.
10. Patient with drug induced severe thrombocytopenia showed complete response after stopping the offending drug
11. Patient with Megaloblastic anaemia showed complete response with supportive management.
12. Steroid with ATT was given in 1 patient of severe thrombocytopenia due to tuberculosis and complete response was achieved while one patient who was given ATT alone showed no response.
13. 12 patients with multifactorial etiology were managed on the lines of underlying etiologies. Complete response was seen in 30% cases, 50% showed partial response and 20% showed no response.

14. One third of the patient with severe thrombocytopenia needed ICU care.
15. Mortality was seen in 5 patients out of whom 3 had sepsis, one patient had dengue shock syndrome and the other one was having autoimmune disease with superadded respiratory infection.

CONCLUSION

Severe thrombocytopenia can have varied presentation which can range from patient being asymptomatic to severe life-threatening bleeding events. Diagnosis of etiology of thrombocytopenia and management of the patient on the lines of underlying etiology is the primary treatment strategy. Platelet transfusions can be given to patients prophylactically in severe thrombocytopenia. Steroids are the mainstay drugs in primary as well as secondary ITP. TPO-RA agents and IVIG are useful adjuncts to steroids in refractory cases.

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Sr. No	MRD NO	INITIALS	AGE	SEX	DATE OF ADMISSION	ETIOLOGY	SUB - ETIOLOGY	ALCOHOLIC HISTORY				H/O TREATMENT (CHEMOTHERAPY, HEPARIN.ABCIXIMAB, QUININE,			COMORBIDITIES	
								YES/ NO	DURATION	AMOUNT	TYPE	YES /NO	DRUG	DURATION OF DRUG GIVEN	TYPE 2 DM	HYPERTENSION
1	1119317	SK	36	F	16-06-2021	SEPSIS	GASTROINTESTINAL	N				N			N	N
2	1121816	MD	70	F	12-06-2021	ITP	-	N	-	-	-	N	-	-	Y	Y
3	1123381	PK	28	F	16-06-2021	SEPSIS	GENITOURINARY	N	-	-	-	N	-	-	N	N
4	1124399	LS	56	M	18-06-2021	SEPSIS	GASTROINTESTINAL	Y	8 YEARS	40MG/DAY	WHISKEY	N	-	-	Y	N
5	1128230	JK	38	F	26-06-2021	ITP	-	N	-	-	-	N	-	-	N	N
6	1128083	MG	26	M	26-06-2021	MULTIFACTORIAL	SKIN AND SOFT TISSUE SEPSIS, TROPICAL FEVER	N	-	-	-	N	-	-	N	N
7	409672	RK	41	F	26-06-2021	AUTOIMMUNE	SLE WITH SECONDARY ITP	N	-	-	-	N	-	-	N	N
8	1142452	B	25	F	25-07-2021	MULTIFACTORIAL	GENITOURINARY SEPSIS, HLH	N	-	-	-	N	-	-	N	N
9	1136497	CS	71	M	13-07-2021	TUBERCULOSIS	-	N	-	-	-	Y	ATT	7MONTHS	N	Y
10	1149111	KK	47	F	06-08-2021	DRUG INDUCED	METHOTREXATE	N	-	-	-	Y	MTX	2 MONTHS	N	Y
11	1150035	GS	90	M	07-08-2021	SEPSIS	GENITOURINARY	N	-	-	-	N	-	-	N	N
12	1128230	JK	38	F	26-06-2021	ITP	-	N	-	-	-	N	-		N	N
13	1178878	KN	62	M	23-09-2021	SEPSIS	RESPIRATORY ,GENITOURINARY	N	-	-	-	N	-	-	Y	Y
14	1184266	RK	48	M	02-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
15	134404	AS	50	M	02-04-2021	TROPICAL FEVER	DENGUE	N	-	-		N	-	-	N	N
16	1185417	RK	18	M	04-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
17	1186424	AK	36	F	06-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
18	1186757	SL	49	M	06-10-2021	ITP	-	N	-	-	-	N	-	-	N	N
19	1186758	MM	25	M	06-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
20	1187237	SK	32	F	07-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
21	1187325	AK	41	F	07-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
22	1187252	SS	56	M	07-10-2021	SEPSIS	GENITOURINARY	N	-	-	-	N	-	-	Y	Y
23	1188058	P	42	F	08-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
24	1188026	VK	71	M	08-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	Y
25	1188077	A	49	M	09-10-2021	MEGALOBLASTIC ANEMIA	-	N	-	-	-	N	-	-	N	N
26	1187959	VB	31	M	08-10-2021	TROPICAL FEVER	-	N	-	-	-	N	-	-	N	N
27	1203197	RG	39	M	07-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
28	1085497	SR	62	F	05-11-2021	MULTIFACTORIAL	DENGUE, ITP	N	-	-	-	N	-	-	Y	Y
29	1203068	NS	18	M	06-11-2022	TROPICAL FEVER	-	N	-	-	-	N	-	-	N	N
30	1203771	AK	21	M	08-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
31	1204168	RA	29	M	08-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
32	1188034	S	36	M	08-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
33	1188709	A	36	M	09-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
34	1188690	RKT	57	M	09-10-2021	TROPICAL FEVER	-	Y	10YEARS	80MG/DAY	WHISKEY	N	-	-	N	N
35	869618	M	24	F	09-10-2021	TROPICAL FEVER	-	N	-	-	-	N	-	-	N	N
36	1188900	NB	18	F	10-10-2021	TROPICAL FEVER	-	N	-	-	-	N	-	-	N	N
37	1189464	JK	35	F	11-10-2021	TROPICAL FEVER	-	N	-	-	-	N	-	-	N	N
38	1189650	PK	28	F	11-10-2021	TROPICAL FEVER	LEPTOSPIRA,DENGUE	N	-	-	-	N	-	-	N	N
39	1189462	IK	44	F	11-10-2021	ITP		N	-	-	-	N	-	-	N	N
40	1188864	KK	51	F	10-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
41	1178280	BK	18	F	22-09-2021	MULTIFACTORIAL	DENGUE,SCRUB,AUTO IMMUNE, HLH	N	-	-	-	N	-	-	N	Y
42	1190408	NK	26	F	13-10-2021	MULTIFACTORIAL	SCRUB , ITP ,AUTO IMMUNE	N	-	-	-	N	-	-	N	N
43	1203102	BB	35	M	06-11-2021	TROPICAL FEVER	DENGUE	Y	4 YEARS	60MG/DAY	WHISKEY	N	-	-	N	N
44	1202172	RS	78	M	05-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	Y	ECOSPRIN	3 YRS	N	Y
45	129688	RKS	50	M	07-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
46	1200362	CB	38	M	31-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
47	1201124	GS	32	M	02-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
48	623569	PS	54	M	01-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	Y	N
49	1115014	MS	34	M	02-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	Y	N
50	1204883	VK	28	M	09-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
51	1204875	A	60	M	09-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
52	1203417	SK	38	F	08-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
53	1204818	AM	29	M	09-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
54	1200451	DC	50	M	31-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-		Y	N

Sr. No	MRD NO								SYMPTOMS/SIGNS							BLEEDING MANIFESTATIONS		
		BRONCHIAL ASTHMA	COAD	CAD	CVA	CKD	THYROID DISEASE	ANY OTHER	FEVER	MALAISE/FATIGUE/GENERALISED BODY WEAKNESS	NAUSEA	VOMITING	DIARRHOEA	CONSTIPATION	BREATHLESSNSS	SUBCONJUNCTIVAL HEMORRHAGE	EPISTAXIS	GUM BLEEDING
1	1119317	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N
2	1121816	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
3	1123381	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	N
4	1124399	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
5	1128230	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y
6	1128083	N	N	N	N	N	N	N	Y	Y	N	N	Y	N	N	N	N	N
7	409672	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
8	1142452	N	N	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	Y
9	1136497	N	N	N	Y	Y	N	N	N	Y	N	N	N	N	N	N	N	N
10	1149111	N	N	N	N	Y	N	RA	N	Y	N	N	Y	N	N	N	N	N
11	1150035	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	N	N
12	1128230	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	N
13	1178878	N	N	N	N	N	N	N	Y	N	N	Y	N	N	Y	N	N	N
14	1184266	N	N	N	N	N	N	N	Y	N	Y	N	N	Y	N	N	N	N
15	134404	N	N	N	N	N	N	N	Y	N	N	Y	N	N	N	N	N	N
16	1185417	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
17	1186424	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
18	1186757	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	Y
19	1186758	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N
20	1187237	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	Y
21	1187325	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	Y
22	1187252	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N
23	1188058	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N
24	1188026	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
25	1188077	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N
26	1187959	N	N	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	N
27	1203197	N	N	N	N	N	N	N	Y	Y	Y	Y	N	N	N	N	N	N
28	1085497	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y
29	1203068	N	N	N	N	N	N	N	Y	N	Y	Y	N	N	N	N	N	N
30	1203771	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
31	1204168	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	N
32	1188034	N	N	N	N	N	N	N	Y	N	N	Y	N	N	N	N	N	N
33	1188709	N	N	N	N	N	N	N	Y	N	Y	Y	N	N	N	N	N	N
34	1188690	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N
35	869618	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
36	1188900	N	N	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	N
37	1189464	N	N	N	N	N	N	N	Y	Y	N	Y	N	N	N	N	N	N
38	1189650	N	N	N	N	N	N	N	Y	N	N	Y	N	N	Y	N	N	Y
39	1189462	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	N	N	Y
40	1188864	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
41	1178280	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
42	1190408	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
43	1203102	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
44	1202172	N	N	N	Y	N	N	N	Y	N	N	Y	N	N	N	N	N	N
45	129688	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
46	1200362	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
47	1201124	N	N	N	N	N	N	N	Y	Y	N	N	N	N	Y	N	N	N
48	623569	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N
49	1115014	N	N	N	N	N	N	N	Y	Y	N	Y	N	N	N	N	N	N
50	1204883	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N
51	1204875	N	Y	N	N	N	N	N	Y	N	Y	Y	N	N	N	N	N	N
52	1203417	N	N	N	N	N	Y	N	Y	N	N	Y	N	N	N	N	N	N
53	1204818	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
54	1200451	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	Y

Sr. No	MRD NO													LAB PARAMETERS			
														TLC (counts/μL)		Hb	
		ECHYMOSES	MALENA	HEMATURIA	MENORRHAGIA	WEIGHT LOSS	ABDOMINAL PAIN	RED SPOTS	RASHES	HEPATOMEGALY	SPLENOMEGALY	LYMPHADENOPATHY	OTHER	TLC (DOA)	TLC (F/U)	Hb (DOA)	Hb(F/U)
1	1119317	N	N	N	N	N	N	N	N	N	Y	N	N	4.1	3.3	9.1	8.4
2	1121816	Y	N	N	N	N	N	N	Y	N	N	N	N	5	10.5	11	11.9
3	1123381	N	N	N	N	N	N	N	N	N	N	N	N	12.4	31.3	10.6	8.4
4	1124399	Y	Y	N	N	N	Y	Y	Y	N	N	N	N	9.7	8.21	8.3	12.8
5	1128230	N	N	N	Y	N	N	N	N	N	N	N	N	3.6	7	6.8	10.3
6	1128083	N	N	N	N	N	N	N	N	N	N	N	N	4	6.3	12	11
7	409672	N	Y	N	Y	N	Y	N	N	N	Y	N	N	6.6	10.3	6.7	11.6
8	1142452	N	N	N	N	N	N	N	N	N	N	N	N	0.7	0.5	6.9	7.6
9	1136497	N	N	N	N	N	N	N	N	N	N	N	N	5.4	2.8	7.8	8.3
10	1149111	N	Y	N	N	N	N	N	N	N	N	N	ORAL ULCERS	0.7	15	10	9
11	1150035	N	N	N	N	N	N	N	N	N	N	N	N	3.2	3	6.1	4.8
12	1128230	N	N	N	Y	N	N	N	N	N	N	N	N	3.6	9.3	6.8	11.1
13	1178878	N	N	N	N	N	Y	N	N	N	N	N	N	5.6	11.7	13	7
14	1184266	N	N	N	N	N	N	N	N	N	N	N	N	3.6	3	17.7	14.8
15	134404	N	N	N	N	N	N	N	N	N	N	N	N	17.8	7.6	13.8	18
16	1185417	N	N	N	N	N	N	N	N	N	N	N	N	5.6	6	14.9	15.1
17	1186424	N	N	N	N	N	Y	N	N	N	N	N	N	11.9	8.5	15.8	10
18	1186757	N	Y	N	N	N	N	N	N	N	N	N	N	8.6	17.3	13.4	12.3
19	1186758	Y	N	N	N	N	N	N	Y	N	Y	N	N	4.6	6.3	18	13
20	1187237	N	Y	N	N	N	N	N	N	N	N	N	N	8	6.3	14.6	12
21	1187325	N	N	N	N	N	N	N	N	N	Y	N	N	7.5	8.1	15	12.3
22	1187252	N	N	N	N	N	N	N	N	N	N	N	N	2.2	-	7.2	-
23	1188058	N	N	N	N	N	N	N	N	N	N	N	N	8.7	7.1	14.8	9.7
24	1188026	N	N	N	N	N	N	N	N	N	N	N	N	2.1	6.3	18	14
25	1188077	N	N	N	N	Y	N	N	N	N	N	N	N	5.2	5.3	8.9	10.7
26	1187959	N	N	N	N	N	N	N	N	N	N	N	N	7.4	7.3	16.8	13.7
27	1203197	N	N	N	N	N	N	N	N	N	N	N	N	12.8	11.7	14.8	12.8
28	1085497	Y	N	N	N	N	N	N	Y	N	N	N	N	11	11.7	9.6	8.7
29	1203068	N	N	N	N	N	N	N	N	N	N	N	N	3.3	5.3	17	14
30	1203771	N	N	N	N	N	N	N	N	N	N	N	N	8.4	5.3	17	15.3
31	1204168	N	N	N	N	N	N	N	N	N	N	N	N	3.7	6.4	15	15.8
32	1188034	N	N	N	N	N	N	N	N	N	Y	N	N	10	6.3	13	12.9
33	1188709	N	Y	N	N	N	N	N	N	N	N	N	N	11.3	7.1	15	11
34	1188690	N	N	N	N	N	N	N	N	N	N	N	N	1.3	6.3	10	11
35	869618	N	N	N	N	N	N	N	N	N	N	N	HEMETEMESIS	3.4	6.3	12.6	10
36	1188900	N	N	N	N	N	Y	N	N	N	N	N	N	4.9	5.3	15	13.9
37	1189464	N	N	N	N	N	N	N	N	N	N	N	N	4.3	7.3	13	13.1
38	1189650	N	N	N	N	N	N	N	N	N	N	N	N	4.6	7.3	9.7	11.3
39	1189462	Y	N	N	N	N	N	Y	Y	N	Y	N	N	8	8.3	12.2	11.9
40	1188864	N	N	N	Y	N	N	N	N	N	N	N	N	4.8	9.3	14.4	11.9
41	1178280	N	N	N	N	N	N	N	N	N	Y	N	N	14.2	7.3	7.7	8.1
42	1190408	Y	N	N	N	N	N	N	Y	N	N	N	N	5.3	11.5	10.9	9.6
43	1203102	N	N	Y	N	N	N	N	N	N	N	N	N	6.8	7.3	17	17
44	1202172	N	N	N	N	N	Y	N	N	N	N	N	N	12.5	7.3	13.1	11
45	129688	N	N	N	N	N	N	N	N	Y	Y	N	N	8.8	11.3	16	13
46	1200362	N	N	N	N	N	N	N	N	N	N	N	N	3	5.3	16	13
47	1201124	N	N	N	N	N	Y	N	N	N	N	N	N	15.8	12.8	19	14
48	623569	N	N	N	N	N	N	N	N	N	N	N	N	4.2	7.3	15	12
49	1115014	Y	N	N	N	N	N	N	Y	Y	N	N	N	7.4	7.3	20	13
50	1204883	N	N	N	N	N	Y	N	N	N	N	N	N	5.1	10.3	13.4	9.8
51	1204875	N	N	N	N	N	N	N	N	N	N	N	N	17.8	8.3	13.8	13.1
52	1203417	N	N	N	N	N	Y	N	N	N	N	N	N	4.5	10.8	14	10
53	1204818	N	N	N	N	N	N	N	N	N	N	N	N	3.3	7.3	15.6	14.1
54	1200451	N	Y	N	N	N	Y	N	N	Y	Y	N	N	7.4	5.3	14	11.6

Sr. No	MRD NO	PLATELETS															LFT								RFT		
		PLT (1)	PLT (2)	PLT (3)	PLT (4)	PLT (5)	PLT (6)	PLT(7)	PLT(8)	PLT(9)	PLT(10)	PLT(15)	PLT(20)	PLT (DISCHARGE/ DAMA/	DAY AT WHICH PLT >30K	PLT(F/U)	BIL (t)	BIL(d)	SGOT	SGPT	ALP	ALBUMIN	TOTAL PROTEINS	UREA	CREATININE	Na
1	1119317	10	22	-	-	-	-	-	-	-	-	-	-	22	-	-	2.08	1.72	24	12	144	2.85	4.7	44	0.58	125
2	1121816	12	14	16	32	52	85	-	-		-	-	-	85	DAY 5	195	0.73	0.18	18	16	82	3/.87	6.6	56	0.72	141
3	1123381	19	22	21	24	48	-	-	-	-	-	-	-	48	DAY 5	-	11.08	8.95	311	163	123	2.4	4.5	127	5.3	143
4	1124399	2	4	6	3	5	4	5	6	8	10	17	142	142	DAY16	275	0.86	0.5	22	38	123	3.06	5	93	1.09	128
5	1128230	10	10	5	6	13	12	28	33	63	94	-	-	142	DAY8	151	0.61	0.17	55	44	64	4	7.9	19	0.57	136
6	1128083	19	22	22	32	40	66	196	-	-	-	-	-	196	DAY3	201	1.8	1.2	94	49	126	3.4	6.1	90	4.09	135
7	409672	19	24	21	20	23	22	25	14	20	44	-	-	59	DAY11	108	1.4	0.98	36	33	57	3.54	6.2	-	-	137
8	1142452	2	14	-	-	-	-		-		-	-	-	14	-	-	1.14	0.42	55	60	97	3.32	6.1	20	0.3	135
9	1136497	19	24	24	30	40	12	18	20	22	22	-	-	9	-	-	2.5	2.2	25	5	139	2.3	4.9	147	3.34	144
10	1149111	12	22	70	83	110	157	-	-	-	-	-	-	157	DAY2	210	0.46	0.1	9	26	209	5.3	5.9	64	1.64	139
11	1150035	14	14	18	16	-	-	-	-	-	-	-	-	16	-	-	1.06	0.35	20	25	107	3.4	65	95	1.7	129
12	1128230	10	10	5	13	12	16	32	74	81	101	-	-	101	DAY7	123	0.61	0.17	55	44	68	4	7.9	19	0.51	136
13	1178878	8	18	40	100	132	149	181	154	150	171	374	421	586	DAY3	620	1.39	0.79	285	166	563	3.5	6.4	11	7.09	119
14	1184266	19	14	18	26	70	147	-	-	-	-	-	-	146	DAY5	162	0.42	0.19	175	144	106	4.3	7.3	22	1.13	136
15	134404	19	57	90	118	-	-	-	-	-	-	-	-	118	DAY2	154	0.85	0.56	286	217	256	3.18	5.8	38	1.11	138
16	1185417	16	44	70	83	144	197	-	-	-	-	-	-	197	DAY2	249	0.85	0.22	232	276	101	3.8	6.7			
17	1186424	16	64	80	70	102	112	-	-	-	-		-	112	DAY2	142	0.36	0.13	254	138	73	2.9	5.5	11	0.62	138
18	1186757	8	2	6	6	8	10	15	14	16	-	-	-	16	-	17	0.48	0.008	34	38	56	3.7	7	30	0.96	140
19	1186758	19	22	36	47	94	-	-	-	-	-	-	-	94	DAY3	154	1.22	0.58	256	174	73	4.08	7	19	0.9	136
20	1187237	10	12	18	24	31	52	-	-	-	-	-	-	52	DAY5	84	0.77	0.32	177	71	125	3.3	5.7	14	0.5	139
21	1187325	18	57	105	143	-	-	-	-	-	-	-	-	143	DAY2	152										
22	1187252	18	-	-	-	-	-	-	-	-	-	-	-	18	-	-										
23	1188058	18	60	64	80	97	119	151	-	-	-	-	-	151	DAY2	194	2.28	1.44	764	310	174	2.9	5.5	21	0.76	136
24	1188026	17	32	32	39	52	82	-	-	-	-	-	-	82	DAY2	107	0.48	0.12	148	91	62	3.8	6.4	21	0.72	131
25	1188077	16	40	30	52	110	-	-	-	-	-	-	-	110	DAY2	156										
26	1187959	8	46	28	48	72	-	-	-	-	-	-	-	72	DAY2	109	0.56	0.17	113	75	73	3.77	6.5	34	1.8	137
27	1203197	19	97	172	-	-	-	-	-	-	-	-	-	172	DAY2	203	0.95		290	160	131	3.4	5.7	21	0.8	135
28	1085497	6	3	6	6	31	62	90	-	-	-	-	-	90	DAY5	153			27	23						
29	1203068	14	35	24	21	50	-	-	-	-	-	-	-	50	DAY2	96	0.37	0.68	1319	606	88	3.7	6.6	22	0.72	127
30	1203771	12	29	55	93	196	-	-	-	-	-	-	-	196	DAY3	214	1.42	0.41	227	144	135	3.92	8	36	0.65	134
31	1204168	19	40	44	72	143	-	-	-	-	-	-	-	143	DAY2	155	0.57	0.09	259	188	115	3.6	6.8	13	0.82	137
32	1188034	19	24	48	80	172	-	-	-	-	-	-	-	172	DAY3	196	0.98	0.3	151	63	733	3.12	6.1	25	0.84	137
33	1188709	10	60	50	54	-	-	-	-	-	-	-	-	54	DAY2	-	3.52	2.49	8518	3388	369	3.2	5.3	59	1.47	131
34	1188690	10	18	45	40	38	61	148	-	-	-	-	-	148	DAY3	196	0.64	0.19	56	75	70	3.3	6.4	32	1.03	140
35	869618	11	26	64	116	119	157	-	-	-	-	-	-	157	DAY3	203	1.52	0.79	3658	838	54			26	0.73	129
36	1188900	19	18	67	100	164	-	-	-	-	-	-	-	164	DAY3	248	0.58	0.19	743	448	61	3.7	6.5	20	0.44	138
37	1189464	19	32	52	98	-	-	-	-	-	-	-	-	98	DAY2	156	0.71	0.16	95	35	111	2.9	5.3	16	0.6	138
38	1189650	8	30	27	15	16	56	48	31	87	121	-	-	12	DAY6	186	1.46	0.93	1205	436	121	2.24	4.9	19	0.49	140
39	1189462	6	12	16	18	76	11.4	163	212	256	233	-	-	233	DAY6	256	0.6	0.1	38	45	123	3.8	7.1	14	0.59	141
40	1188864	19	18	28	43	78	95	-	-	-	-	-	-	95	DAY4	139	1.35	1	410	217	261	2.6	4.9	13	0.43	141
41	1178280	19	31	46	10	18	30	24	60	80	110	58	50	50	DAY2	-	1.23	0.51	191	91	207	2.5	6	77	0.98	143
42	1190408	14	60	57	104	141	190	-	-	-	-	-	-	190	DAY2	210	1.41	0.98	31	16	90	4.5	9.1	12	0.55	139
43	1203102	8	16	28	30	33	75	-	-	-	-	-	-	75	DAY5	101	0.5	0.12	237	157	164	3.6	6.7	30	0.87	140
44	1202172	19	25	24	28	62	94	140	146	180	270	-	-	273	DAY 5	286	0.5	0.16	57	25	69	2.8	6.2	24	0.9	134
45	129688	18	20	82	139	-	-	-	-	-	-	-	-	139	DAY3	156			242	140						
46	1200362	19	25	28	40	61	-	-	-	-	-	-	-	61	DAY4	96	0.76	0.42	411	189	220	3.62	6.9	17	0.89	134
47	1201124	9	55	57	63	104	126	174	182	-	-	-	-	182	DAY2	196	1.88		2294	1469						
48	623569	19	34	41	55	-	-	-	-	-	-	-	-	55	DAY2	96	0.65	0.24	150	105	190	4.19	7.4	15	0.91	134
49	1115014	19	14	48	101	-	-	-	-	-	-	-	-	101	DAY3	156	0.61	0.31	198	189	101	3.1	6	20	0.68	134
50	1204883	12	35	36	64	119	-	-	-	-	-	-	-	119	DAY2	156	2.35	1.95	646	387	300	3.17	5.2	13	0.67	137
51	1204875	19	57	90	118	-	-	-	-	-	-	-	-	118	DAY2	159	0.85	0.56	286	217	255	3.18	5.8	38	1.11	138
52	1203417	19	30	36	43	-	-	-	-	-	-	-	-	43	DAY2	-	0.44	0.23	394	179	98	3.2	6.2	15	0.82	137
53	1204818	16	24	24	100	-	-	-	-	-	-	-	-	100	DAY4	159	0.93	0.29	100	62	159	3.1	6.8	25	0.8	137
54	1200451	10	18	64	73	58	44	50	28	32	66	-	-	166	DAY3	192	2.38	1.68	675	323	300	2.6	5.7	62	1.22	145

Sr. No	MRD NO			PBF	BONE MARROW				AUTOIMMUNE PROFILE		VIRAL MARKERS
		K	Cl		IF DONE	CELLULARITY	MEGAKARYOCYTES	OTHER	ANA	APLA	
1	1119317	3.87	101	NCNC	N	-	-	-	-	-	NR
2	1121816	4.4	101	NCNC	Y	HYPER	Y	ITP	-	-	NR
3	1123381	5.09	109	MCHC	N	-	-	-	-	-	NR
4	1124399	6.26	92	NCNC	Y	NORMO	N	ERYTHROID PROMINENCE	NEG	-	NR
5	1128230	3.67	107	NCNC	Y	HYPER	Y	ITP WITH IDA	POS(SSA/RO52)	-	NR
6	1128083	4.67	99	NCNC	N	-	-	-	-	-	NR
7	409672	3.8	102	MCHC +TEAR DROP CELLS +SCISTOCYTES	Y	HYPER	Y	ITP	POS(SSA/RO52)	-	NR
8	1142452	4.13	104	MCHC	Y	HYPER	N	HEMOPHAGOCYTOSIS	-	-	NR
9	1136497	2.9	109	MCHC	Y	NORMO	N	GRANULOMATOUS INFLAMMATION	NEG	-	NR
10	1149111	3.56	107	NCNC	N	-	-	-	-	-	NR
11	1150035	4.8	102	NCNC	Y	HYPER	N	ERYTHROID PROMINENCE	-	-	NR
12	1128230	3.61	107	NCNC	Y	HYPER	N	ITP	POS(SSA/RO52)	-	NR
13	1178878	5.33	93	NCNC	N	-	-	-	-	-	NR
14	1184266	5.29	99	NCNC	N	-	-	-	-	-	NR
15	134404	5.18	108	-	N	-	-	-	-	-	NR
16	1185417			NCNC	N	-	-	-	-	-	NR
17	1186424	3.82	106	NCNC	N	-	-	-	-	-	NR
18	1186757	3.96	106	NCNC	Y	HYPER	N	ITP	-	-	NR
19	1186758	4.16	99	NCNC	N	-	-	-	-	-	NR
20	1187237	3.67	103	NCNC	N	-	-	-	-	-	NR
21	1187325			NCNC	N	-	-	-	-	-	NR
22	1187252			-	N	-	-	-	-	-	NR
23	1188058	5.8	101	NCNC	N	-	-	-	-	-	NR
24	1188026	4.41	94	NCNC	N	-	-	-	-	-	NR
25	1188077			MACROCYTES SEEN	Y	HYPER	Y	MEGALOBLASTIC ANEMIA	NEG	-	NR
26	1187959	4.27	106	NCNC	N	-	-	-	2+ PROFILE-	-	NR
27	1203197	3.5	96	NCNC	N	-	-	-	-	-	NR
28	1085497			NCNC	Y	HYPER	Y	ITP	-	-	NR
29	1203068	5.2	94	NCNC	N	-	-	-	-	-	NR
30	1203771	4.6	98	NCNC	N	-	-	-	-	-	NR
31	1204168	4.6	102	NCNC	N	-	-	-	-	-	NR
32	1188034	4.73	105	NCNC	N	-	-	-	-	-	NR
33	1188709	7.4	90	NCNC	N	-	-	-	-	-	NR
34	1188690	4.7	107	ANISOCYTOSIS,MACROCYTES,NC	Y	HYPER	N	ERYTHROID PROMINENCE	NEG	-	NR
35	869618	4	98	NCNC	N	-	-	-	3+PROFILE-	-	NR
36	1188900	4.62	107	NCNC	N	-	-	-	-	-	NR
37	1189464	4.32	105	NCNC	N	-	-	-	-	-	NR
38	1189650	3.16	104	NCNC	N	-	-	-	-	-	NR
39	1189462	4.21	105	NCNC	Y	HYPER	N	ITP	2+ PROFILE-	-	NR
40	1188864	4.07	107	NCNC	N	-	-	-	-	-	NR
41	1178280	4.5	104	NCNC	Y	NORMO	N	HEMOPHAGOCYTOSIS	4+ KU/UCTD	-	NR
42	1190408	4.4	102	NCNC	Y	HYPER	N	ITP	4+ SSA/RP60+ SSB/LA	-	NR
43	1203102	4.08	106	NCNC	N	-	-	-	-	-	NR
44	1202172	4.8	100	NCNC	N	-	-	-	-	-	NR
45	129688			NCNC	N	-	-	-	-	-	NR
46	1200362	4.8	99	NCNC	N	-	-	-	-	-	NR
47	1201124			NCNC	N	-	-	-	-	-	NR
48	623569	5	100	NCNC	N	-	-	-	-	-	NR
49	1115014	4.7	99	NCNC	N	-	-	-	-	-	NR
50	1204883	5.08	107	NCNC	N	-	-	-	-	-	NR
51	1204875	5.18	108	-	N	-	-	-	-	-	NR
52	1203417	5	100	NCNC	N	-	-	-	-	-	NR
53	1204818	4.7	101	NCNC	N	-	-	-	-	-	NR
54	1200451	4.5	115	NCNC	N	-	-	-	-	-	NR

Sr. No	MRD NO	TROPICAL FEVER	D DIMER	PROTHROMBIN TIME	FIBRINOGEN	RADIOLOGICAL INVESTIGATION
						USG WHOLE ABDOMEN
1	1119317	-	-	16.8	-	CIRRHOSSIS WITH PORTAL HYPERTENSION
2	1121816	NEGATIVE	0.43	12.4	1.14	FATTY LIVER GRADE 1
3	1123381	NEGATIVE	64	16.4	313	ACUTE MEDICAL RENAL DISEASE
4	1124399	NEGATIVE	-	14.3	-	LIVER PARENCHYMAL CHANGES
5	1128230	NEGATIVE	-	11.3	-	FATTY LIVER GRADE 1
6	1128083	NEGATIVE	-	15.1	-	ACUTE MEDICAL RENAL DISEASE
7	409672	NEGATIVE	-	12.2	-	SPLENOMEGALY
8	1142452	NEGATIVE	-	11.4	-	NAD
9	1136497	-	-	12.7	-	LIVER PARENCHYMAL CHANGES
10	1149111	NEGATIVE	-	12.2	-	BILATERAL SMALL KIDNEYS
11	1150035	NEGATIVE	0.79	16.6	-	-
12	1128230	-	-	12.1	-	-
13	1178878	NEGATIVE	-	9.7	-	FATTY LIVER GRADE 2 ,ACUTE MEDICAL RENAL DS
14	1184266	DENGUE NS1		11.2	-	REACTIVE GB WALL EDEMA
15	134404	DENGUENS1	-	9.6	-	FATTY LIVER GRADE 1
16	1185417	DENGUE IGM	-	10.1	-	MILD HEPATOMEGALY
17	1186424	DENGUE NS1	-	11.2	-	FATTY LIVER GRADE 1,REACTIVE GALL BLADDER WALL EDEMA
18	1186757	NEGATIVE	-	10.2	0.94	FATTY LIVER GRADE 1
19	1186758	-	-	10.7	-	SPLENOMEGALY
20	1187237	DENGUE IGM	-	11.6	-	EDEMATPUS GALL BLADDER .B/L PLEURAL EFFUSION ,MINIMAL ASCITES
21	1187325	DENGUE IGM	-	10.1	-	SPLENOMEGALY
22	1187252	-	-	11.1	-	-
23	1188058	DENGUE NS1	-	12.6	-	MILD SPLENOMEGALY
24	1188026	DENGUE NS1	-	10.9	-	FATTY LIVER GRADE 1
25	1188077	-	-	10.1	-	NAD
26	1187959	NEGATIVE	-	12.3	-	REACTIVE GB WALL EDEMA
27	1203197	DENGUE IGM	-	11.2	-	FATTY LIVER GRADE 2 ,SPLENOMEGALY
28	1085497	DENGUE IGM	-	10.2	-	NAD
29	1203068	NEGATIVE	-	13.1	-	MILD SPLENOMEGALY
30	1203771	DENGUENS1	-	11.3	-	NAD
31	1204168	DENGUENS1	-	10.2	-	-
32	1188034	DENGUENS1	-	12	-	BORDERLINE SPLENOMEGALY
33	1188709	DENGUENS1	-	25	-	REACTIVE GB WALL EDEMA
34	1188690	NEGATIVE	-	11.6	-	-
35	869618	NEGATIVE	-	11.3	-	REACTIVE GB WALL EDEMA
36	1188900	NEGATIVE	-	11.5	-	FATTY LIVER GRADE 1
37	1189464	NEGATIVE	-	10.4	-	FATTY LIVER GRADE 1,REACTIVE GALL BLADDER WALL EDEMA
38	1189650	DENGUE IGM,LEPTOSPIRA IGM	-	10.5	-	SINGLE LIVE INTRAUTERINE FETUS WITH GESTATIONAL AGE OF 31 WEEKS
39	1189462	NEGATIVE	-	10.8	-	FATTY LIVER GRADE 1 ,SPLENOMEGALY
40	1188864	DENGUE NS1	-	12.1	-	FATTY LIVER GRADE 2,REACTIVE GALL BLADDER WALL EDEMA
41	1178280	DENGUE IGM,SCRUB IGM	-	12.2	-	FATTY LIVER GRADE 2 ,SPLENOMEGALY
42	1190408	SCRUB IGM	-	13.8	-	NAD
43	1203102	DENGUE NS1	-	10.9	-	REACTIVE GB WALL EDEMA
44	1202172	DENGUE NS1	-	11.9	-	MILD ASCITES
45	129688	DENGUE NS1	-	13.6	-	MILD HEPATOSPLENOMEGALY,CHRONIC PANCREATITIS
46	1200362	DENGUE NS1	-	11.8	-	-
47	1201124	DENGUE IGM	-	12.1	-	GROSS ASCITES WITH B/L PLEURAL EFFUSION
48	623569	DENGUE IGM	-	11.1	-	-
49	1115014	DENGUE IGM	-	10.7	-	FATTY LIVER GRADE 1
50	1204883	DENGUE NS1	-	17.9	-	FATTY LIVER GRADE 2,MILD ASCITES ,MILD B/L PLEURAL EFFUSION
51	1204875	DENGUE NS1	-	9.6	-	FATTY LIVER GRADE 2,MILD ASCITES,REACTIVE GB WALL EDEMA
52	1203417	DENGUE NS1	-	10.9	-	REACTIVE GB WALL EDEMA ,B/L PLEURAL EFFUSION,MODERATE ASCITES,FATTY LIVER GRADE 1
53	1204818	DENGUE IGM	-	11	-	NAD
54	1200451	DENGUE IGM	-	13.8	-	HEPATOSPLENOMEGALY

Sr. No	MRD NO	OTHER CT	RDP	SDP	RDP + SDP	INTERVENTION							
						RDP	SDP	STEROIDS			TPO AGONIST		
								DRUG	DOSE	DURATION	DRUG USED	DOSE	DURATION
1	1119317	CIRRHOSSIS WITH PORTAL HTN AND GROSS ASCITES ,PORTAL VEIN AND SPLENIC VEIN THROMBOSIS	Y	N	Y	2	-	-	-	-	-	-	-
2	1121816	-	N	N	N	-	-	METHYLPREDNISOLONE	500MG	5D	-	-	-
3	1123381	-	N	N	N	-	-	-	-		-	-	-
4	1124399	-	Y	Y	Y	2	10	DEXAMETHASONE	4MG	7D	ROMIPLOSTIM	250MCG	1D
5	1128230	-	Y	Y	Y	4	5	DEXAMETHASONE	4MG	6D	-	-	-
6	1128083	-	N	N	N	-	-	DEXAMETHASONE	8MG	1D	-	-	-
7	409672	-	Y	N	N	3	-	METHYLPREDNISOLONE	1000MG	3D	-	-	-
8	1142452	-	N	Y	N	-	1	METHYLPREDNISOLONE	500MG	2D	-	-	-
9	1136497	-	N	N	N	-	-	DEXAMETHASONE	8MG	14D	ROMIPLOSTIM	300MCG	2D
10	1149111	-	N	N	N	-	-	-	-	-	FILGRASTIM	300MCG	1D
11	1150035	-	N	Y	N	-	1	-	-	-	-	-	-
12	1128230	-	Y	Y	Y	9	1	METHYLPREDNISOLONE	1000MG	3D	-	-	-
13	1178878	B/L CONSOLIDATION AND GGO IN LUNGS	N	Y	N	-	1	HYROCORTISONE	150MG	10D	-	-	-
14	1184266	-	N	Y	N	-	1	-	-	-	-	-	-
15	134404	-	N	Y	N	-	1	-	-	-	-	-	-
16	1185417	-	Y	N	N	2	-	-	-	-	-	-	-
17	1186424	-	Y	N	N	4	-	-	-	-	-	-	-
18	1186757	-	N	N	N	-	-	DEXAMETHASONE	8MG	3D	ELTROMOBOPAG	50MG	4D
19	1186758	-	N	Y	N	-	1	-	-	-	-	-	-
20	1187237	-	Y	Y	Y	2	2	-	-	-	-	-	-
21	1187325	-	N	Y	N	-	1	-	-	-	-	-	-
22	1187252	-	N	N	N	-	-	-	-	-	-	-	
23	1188058	-	N	Y	N	-	1	-	-	-	-	-	-
24	1188026	-	N	Y	N	-	1	-	-	-	-	-	-
25	1188077	-	Y	N	N	2	-	-	-	-	-	-	-
26	1187959	-	N	Y	N	-	2	-	-	-	-	-	-
27	1203197	-	N	Y	N	-	1	-	-	-	-	-	-
28	1085497	-	N	Y	N	-	2	DEXAMETHASONE	40MG	3D	-	-	-
29	1203068	-	Y	Y	Y	2	1	-	-	-	-	-	-
30	1203771	-	N	Y	N	-	1	-	-	-	-	-	-
31	1204168	-	N	Y	N	-	1	-	-	-	-	-	-
32	1188034	-	N	N	N	-	-	-	-	-	-	-	-
33	1188709	-	N	Y	N	-	1	-	-	-	-	-	-
34	1188690	COPD ,EXTENSIVE FIBROCALCIFIED AND FIBROCYSTIC LESION IN B/L LUNGS	Y	Y	Y	2	1	-	-	-	-	-	-
35	869618	-	N	Y	N	-	1	-	-	-	-	-	-
36	1188900	-	Y	N	N	2	-	-	-	-	-	-	-
37	1189464	-	N	N	N	-	-	-	-	-	-	-	-
38	1189650	-	Y	Y	Y	4	1	-	-	-	-	-	-
39	1189462	-	Y	Y	Y	4	5	DEXAMETHASONE	40MG	3D	-	-	-
40	1188864	-	Y	N	N	2	-	-	-	-	-	-	-
41	1178280	MRI-VASCULITIC INFARCTS	N	N	N	-	-	METHYLPREDNISOLONE	1G	3D	-	-	-
42	1190408	-	N	N	N	-	-	METHYLPREDNISOLONE	1G	3D	-	-	-
43	1203102	-	N	N	N	-	-	-	-	-	-	-	-
44	1202172	CTWA-SMALL AND LARGE BOWEL DIVERTICULOSIS WITH DIVERTICULITIS	N	N	N	-	-	-	-	-	-	-	-
45	129688	-	N	Y	N	-	1	-	-	-	-	-	-
46	1200362	-	N	Y	N	-	1	-	-	-	-	-	-
47	1201124	-	N	Y	N	-	1	-	-	-	-	-	-
48	623569	-	Y	N	N	2	-	-	-	-	-	-	-
49	1115014	-	N	Y	N	-	1	-	-	-	-	-	-
50	1204883	-	N	Y	N	-	1	-	-	-	-	-	-
51	1204875	-	N	Y	N	-	1	-	-	-	-	-	-
52	1203417	-	N	Y	N	-	2	-	-	-	-	-	-
53	1204818	-	N	Y	N	-	1	-	-	-	-	-	-
54	1200451	-	Y	Y	Y	2	2	-	-	-	-	-	-

Sr. No	MRD NO	OUTCOMES								
		OTHER INTERVENTION (IVIG/SPLENECTOMY	COMPLETE	PARTIAL	NO RESPONSE	LENGTH OF HOSPITAL STAY	NEED FOR ICU CARE	NEED FOR		DISCHARGE /DAMA/ MORTALITY
								Y/N	DURATION	
1	1119317	-	-	-	Y	2D	Y	Y	-	MORTALITY
2	1121816	-	Y	-	-	6D	N	N	-	DISCHARGE
3	1123381	-	-	Y	-	5D	Y	Y	5D	MORTALITY
4	1124399	IVIG	Y	-	-	20D	N	N	-	DISCHARGE
5	1128230	-	Y	-	-	11D	N	N	-	DISCHARGE
6	1128083	-	Y	-	-	7D	N	N	-	DISCHARGE
7	409672	-	Y	-	-	14D	Y	N	-	DISCHARGE
8	1142452	-	-	-	-	2D	Y	N	1D	DAMA
9	1136497	-	-	-	-	14D	Y	N	-	DAMA
10	1149111	-	Y	-	-	6D	N	N	-	DISCHARGE
11	1150035	-	-	-	-	4D	N	N	-	DAMA
12	1128230	-	Y	-	-	11D	N	N	-	DISCHARGE
13	1178878	-	Y	-	-	21D	Y	Y	10D	DISCHARGE
14	1184266	-	Y	-	-	6D	N	N	-	DISCHARGE
15	134404	-	Y	-	-	4D	N	N	-	DISCHARGE
16	1185417	-	Y	-	-	6D	N	N	-	DISCHARGE
17	1186424	-	Y	-	-	6D	N	N	-	DISCHARGE
18	1186757	-	-	-	Y	9D	N	N	-	DISCHARGE
19	1186758	-	Y	-	-	5D	N	N	-	DISCHARGE
20	1187237	-	-	Y	-	6D	N	N	-	DISCHARGE
21	1187325	-	Y	-	-	4D	N	N	-	DISCHARGE
22	1187252	-	-	-	-	1D	N	N	-	DAMA
23	1188058	-	Y	-	-	7D	N	N	-	DISCHARGE
24	1188026	-	Y	-	-	6D	N	N	-	DISCHARGE
25	1188077	-	Y	-	-	5D	N	N	-	DISCHARGE
26	1187959	-	Y	-	-	5D	N	N	-	DISCHARGE
27	1203197	-	Y	-	-	3D	N	N	-	DISCHARGE
28	1085497	-	Y	-	-	6D	N	N	-	DISCHARGE
29	1203068	-	-	Y	-	5D	N	N	-	DISCHARGE
30	1203771	-	Y	-	-	5D	N	N	-	DISCHARGE
31	1204168	-	Y	-	-	5D	N	N	-	DISCHARGE
32	1188034	-	Y	-	-	5D	N	N	-	DISCHARGE
33	1188709	-	-	Y	-	4D	Y	Y	3D	MORTALITY
34	1188690	-	Y	-	-	7D	N	N	-	DISCHARGE
35	869618	-	Y	-	-	6D	N	N	-	DISCHARGE
36	1188900	-	Y	-	-	5D	N	N	-	DISCHARGE
37	1189464	-	Y	-	-	4D	N	N	-	DISCHARGE
38	1189650	-	Y	-	-	14D	Y	N	-	DISCHARGE
39	1189462	-	Y	-	-	10D	N	N	-	DISCHARGE
40	1188864	-	Y	-	-	6D	N	N	-	DISCHARGE
41	1178280	-	-	Y	-	20D	N	Y	-	DAMA
42	1190408	-	Y	-	-	6D	N	N	-	DISCHARGE
43	1203102	-	Y	-	-	6D	N	N	-	DISCHARGE
44	1202172	-	Y	-	-	11D	Y	N	-	DISCHARGE
45	129688	-	Y	-	-	4D	N	N	-	DISCHARGE
46	1200362	-	-	Y	-	5D	N	N	-	DISCHARGE
47	1201124	-	Y	-	-	8D	N	N	-	DISCHARGE
48	623569	-	-	Y	-	4D	N	N	-	DISCHARGE
49	1115014	-	Y	-	-	4D	N	N	-	DISCHARGE
50	1204883	-	Y	-	-	5D	N	N	-	DISCHARGE
51	1204875	-	Y	-	-	5D	N	N	-	DISCHARGE
52	1203417	-	-	Y	-	4D	N	N	-	DAMA
53	1204818	-	Y	-	-	4D	N	N	-	DISCHARGE
54	1200451	-	Y	-	-	14D	Y	N	-	DISCHARGE

Sr. No	MRD NO	INITIALS	AGE	SEX	DATE OF ADMISSION	ETIOLOGY	SUB - ETIOLOGY	ALCOHOLIC HISTORY				H/O TREATMENT (CHEMOTHERAPY, HEPARIN.ABCIXIMAB, QUININE,			COMORBIDITIES	
								YES/ NO	DURATION	AMOUNT	TYPE	YES /NO	DRUG	DURATION OF DRUG GIVEN	TYPE 2 DM	HYPERTENSION
55	1203614	ZH	23	M	08-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
56	1203172	CS	70	F	06-11-2021	AUTOIMMUNE	-	N	-	-	-	N	-	-	N	N
57	1204193	AK	32	M	08-11-2021	ITP	-	N	-	-	-	N	-	-	N	N
58	119338	SK	44	M	18-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	Y	N
59	1193870	MK	58	F	19-10-2021	TROPICAL FEVER	LEPTOSPIRA	N	-	-	-	N	-	-	N	Y
60	1196407	PK	60	F	23-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	Y	Y
61	1042338	PK	61	F	05-03-2022	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
62	1138743	GK	40	F	17-07-2021	MULTIFACTORIAL	DENGUE, ITP	N	-	-	-	N	-	-	N	N
63	1138761	JK	68	F	17-09-2021	TROPICAL FEVER	-	N	-	-	-	N	-	-	Y	Y
64	1135959	RS	23	M	12-07-2021	ITP	-	N	-	-	-	N	-	-	N	N
65	1018578	RG	41	M	16-07-2021	MULTIFACTORIAL	LEPTOSPIRA, ITP	N	-	-	-	N	-	-	N	N
66	260600	TS	49	M	25-07-2021	ITP	-	N	-	-	-	N	-	-	N	N
67	432189	RK	66	M	10-10-2021	SEPSIS	GASTROINTESTINAL	Y	14 YEARS	100MG/DAY	WHISKEY	N	-	-	Y	Y
68	1138743	GK	40	F	17-07-2021	MULTIFACTORIAL	DENGUE, ITP	N	-	-	-	N	-	-	N	N
69	1138761	JK	68	F	17-07-2021	TROPICAL FEVER	-	N	-	-	-	N	-	-	Y	Y
70	1140878	DK	58	M	27-07-2021	TUBERCULOSIS	-	N	-	-	-	N	-	-	Y	N
71	1143678	RS	34	M	27-07-2021	ITP	-	N	-	-	-	N	-	-	N	N
72	1128230	JK	38	F	26-06-2021	MULTIFACTORIAL	-	N	-	-	-	N	-	-	N	N
73	1120956	PK	21	F	10-06-2021	TROPICAL FEVER	DENGUE,MALARIA	N	-	-	-	N	-	-	N	N
74	1121162	MK	30	F	11-06-2021	SEPSIS	GENITOURINARY	N	-	-	-	N	-	-	N	N
75	1122909	HC	56	M	15-06-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	Y	N
76	1112819	RK	43	M	24-05-2021	ITP	-	N	-	-	-	N	-	-	Y	N
77	1110990	AM	19	M	20-05-2021	ITP	-	N	-	-	-	N	-	-	N	N
78	1101303	GD	52	F	11-05-2021	ITP	-	N	-	-	-	N	-	-	N	N
79	1090639	RD	78	F	10-04-2021	TROPICAL FEVER	LEPTOSPIRA	N	-	-	-	N	-	-	Y	Y
80	1086931	JS	49	M	15-04-2021	SEPSIS	GENITOURINARY	Y	25 YEARS	40MG/DAY	WHISKEY	N	-	-	Y	N
81	1079522	A	19	M	07-04-2021	TROPICAL FEVER	LEPTOSPIRA,ENTERIC	N	-	-	-	N	-	-	N	N
82	1071064	DK	28	F	31-03-2021	ITP	-	N	-	-	-	N	-	-	N	N
83	1030661	JS	36	M	18-02-2021	ITP	-	Y	10YEARS	80MG/DAY	WHSIKEY	N	-	-	N	N
84	1308835	S	25	F	22-05-2021	SEPSIS	-	N	-	-	-	N	-	-	N	N
85	1295666	SB	68	M	29-04-2021	SEPSIS	-	N	-	-	-	Y	TACROLIMUS,MMF	20 YEARS	Y	N
86	1311078	MD	50	M	25-05-2021	ITP	-	N	-	-	-	N	-	-	N	N
87	1313325	JS	82	M	30-05-2021	MULTIFACTORIAL	GENITOURINARY SEPSIS, HLH	N	-	-	-	N	-	-	Y	N
88	1315554	SK	45	M	02-06-2021	SEPSIS	GASTROINTESTINAL	N	-	-	-	N	-	—	Y	N
89	1106578	MSB	78	M	08-06-2021	ITP	-	N	-	-	-	N	-	-	Y	Y
90	1318508	AS	52	M	13-06-2021	SEPSIS	GENITOURINARY	Y	20 YEARS	40MG/DAY	WHISKEY	N	-	-	N	N
91	1293775	N	67	F	26-04-2021	MEGALOBLASTIC ANEMIA	-	N	-	-	-	N	-	-	N	N
92	1302433	AD	57	M	12-05-2021	SEPSIS	GENITOURINARY	Y	15 YEARS	60MG/DAY	WHISKEY	N	-	-	Y	Y
93	412900	JS	59	M	12-05-2021	SEPSIS	RESPIRATORY	Y	15 YEARS	60MG/DAY	WHISKEY	N	-	-	N	N
94	1302993	CK	23	M	12-05-2021	TROPICAL FEVER	-	N	-	-	-	N	-	-	N	N
95	1270231	KD	73	F	12-05-2021	MULTIFACTORIAL	RESPIRATORY SEPSIS,AUTO IMMUNE	N	-	-	-	N	-	-	N	Y
96	1303493	BS	65	M	13-05-2021	TROPICAL FEVER	-	N	-	-	-	N	-	-	N	N
97	1296993	BS	80	M	13-05-2021	SEPSIS	GENITOURINARY	Y	25 YEARS	40MG/DAY	WHISKEY	N	-	-	Y	Y
98	1295978	N	63	F	14-05-2021	SEPSIS	GASTROINTESTINAL	Y	20 YEARS	80MG/DAY	WHISKEY	N	-	-	N	Y
99	1305231	SD	50	F	17-05-2021	ITP	-	N	-	-	-	N	-	-	N	N
100	1306683	NK	22	M	19-09-2021	SEPSIS	GASTROINTESTINAL	N	-	-	-	N	-	-	N	N
101	1305964	NS	23	M	20-05-2021	SEPSIS	INFECTIVE ENDOCARDITIS	N	-	-	-	N	-	-	N	N
102	1307311	H	25	F	19-05-2021	SEPSIS	RESPRATORY,GENITOURINARY	N	-	-	-	N	-	-	N	N
103	1306643	R	19	M	21-05-2021	SEPSIS	GENITOURINARY	N	-	-	-	N	-	-	N	N
104	1316162	HK	35	F	02-06-2021	SEPSIS	GASTROINTESTINAL	N	-	-	-	N	-	-	N	N
105	1245032	SK	33	F	03-06-2021	AUTOIMMUNE	SLE WITH SECONDARY ITP	N	-	-	-	N	-	-	N	N
106	1318459	VK	63	M	06-06-2021	TUBERCULOSIS	-	N	-	-	-	N	-	-	Y	N
107	1318429	L	46	M	02-02-1900	SEPSIS	GASTROINTESTINAL	Y	20 YEARS	60MG/DAY	WHISKEY	N	-	-	Y	Y
108	1399640	I	45	F	18-10-2021	TROPICAL FEVER	-	N	-	-	-	N	-	-	Y	N

Sr. No	MRD NO								SYMPTOMS/SIGNS									
		BRONCHIAL ASTHMA	COAD	CAD	CVA	CKD	THYROID DISEASE	ANY OTHER	FEVER	MALAISE/FATIGUE/GENERALISED BODY WEAKNESS	NAUSEA	VOMITING	DIARRHOEA	CONSTIPATION	BREATHLESSNSS	BLEEDING MANIFESTATIONS		
																SUBCONJUNCTIVAL HEMORRHAGE	EPISTAXIS	GUM BLEEDING
55	1203614	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
56	1203172	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
57	1204193	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
58	119338	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N
59	1193870	N	N	N	N	N	N	N	Y	N	N	Y	Y	N	N	N	N	N
60	1196407	N	N	N	N	N	Y	N	Y	Y	N	N	N	N	N	N	N	N
61	1042338	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	Y
62	1138743	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N
63	1138761	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N
64	1135959	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
65	1018578	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y
66	260600	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y
67	432189	N	N	N	N	N	N	N	Y	N	Y	Y	N	N	N	N	N	N
68	1138743	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
69	1138761	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N
70	1140878	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N
71	1143678	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N
72	1128230	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	N
73	1120956	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N
74	1121162	N	N	N	N	N	N	N	Y	N	N	Y	N	N	Y	N	N	N
75	1122909	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
76	1112819	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N
77	1110990	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
78	1101303	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N
79	1090639	N	N	Y	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N
80	1086931	N	N	N	N	Y	N	N	Y	Y	N	N	N	N	Y	N	N	N
81	1079522	N	N	N	N	N	N	N	Y	N	N	N	Y	N	N	N	N	N
82	1071064	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	Y
83	1030661	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N
84	1308835	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	N	N
85	1295666	N	N	Y	N	Y	N	N	Y	Y	N	N	N	N	N	N	N	N
86	1311078	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y
87	1313325	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N
88	1315554	N	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	N	N	N
89	1106578	N	N	Y	N	Y	N	N	N	N	N	Y	N	N	N	N	N	N
90	1318508	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	N
91	1293775	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	N	N
92	1302433	N	N	N	N	Y	N	N	Y	Y	N	N	N	N	N	N	N	N
93	412900	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	N
94	1302993	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	Y	N
95	1270231	N	N	Y	N	N	N	N	Y	Y	Y	N	N	N	Y	Y	N	N
96	1303493	N	N	N	N	N	N	N	Y	Y	Y	Y	N	N	N	N	N	N
97	1296993	N	N	N	N	N	N	CLD	Y	Y	N	N	N	Y	Y	N	N	N
98	1295978	N	N	Y	N	N	Y	CLD	Y	Y	N	Y	Y	N	N	N	N	N
99	1305231	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N
100	1306683	N	N	N	N	N	N	N	Y	N	N	N	Y	N	N	N	N	N
101	1305964	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N
102	1307311	N	N	N	N	N	N	N	Y	Y	N	N	N	N	Y	Y	N	N
103	1306643	N	N	N	N	N	N	N	Y	Y	N	N	N	N	Y	N	N	N
104	1316162	N	N	N	N	N	N	N	Y	Y	N	Y	N	N	Y	N	N	Y
105	1245032	N	N	N	N	N	N	N	Y	N	Y	N	N	N	Y	N	N	N
106	1318459	N	N	N	N	N	N	N	Y	Y	N	N	N	N	Y	N	N	N
107	1318429	N	N	N	N	N	N	N	Y	Y	Y	Y	N	N	Y	N	N	N
108	1399640	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N

Sr. No	MRD NO													LAB PARAMETERS			
														TLC (counts/μL)		Hb	
		ECHYMOSES	MALENA	HEMATURIA	MENORRHAGIA	WEIGHT LOSS	ABDOMINAL PAIN	RED SPOTS	RASHES	HEPATOMEGALY	SPLENOMEGALY	LYMPHADENOPATHY	OTHER	TLC (DOA)	TLC (F/U)	Hb (DOA)	Hb(F/U)
55	1203614	N	Y	N	N	N	N	N	N	N	N	N	N	7.7	5.5	16	13.5
56	1203172	N	Y	N	N	N	N	N	N	N	N	N	HEMETEMESIS,DRYMOUTH	4.5	10.8	17	10.9
57	1204193	N	Y	N	N	N	Y	N	N	N	N	N	N	8.7	9.5	4.4	8
58	119338	N	N	N	N	N	N	N	N	N	N	N	N	3.4	7.4	18.6	14.6
59	1193870	N	N	N	N	N	Y	N	N	N	N	N	N	6.2	6.6	11.5	8.2
60	1196407	N	N	N	N	N	N	N	N	N	N	N	N	4.3	8.9	13.6	11.6
61	1042338	N	N	N	N	N	N	N	N	N	N	N	N	14	13	11	11.8
62	1138743	Y	N	N	N	N	Y	N	Y	N	N	N	N	11	13	11.5	10.9
63	1138761	N	N	N	N	N	N	N	N	N	N	N	N	15.2	12	12	9
64	1135959	N	N	N	N	N	N	Y	N	N	N	N	N	17.2	17.8	13.7	13
65	1018578	N	N	N	N	N	N	N	N	N	N	N	N	7.8	14.1	12.3	11
66	260600	N	N	N	N	N	N	N	N	N	N	N	N	9.8	13.8	17	15.7
67	432189	N	N	N	N	N	N	N	N	N	N	N	N	1.9	1.7	3.6	7.1
68	1138743	Y	N	N	N	N	Y	N	Y	N	N	N	N	11.5	13	11.5	10.9
69	1138761	N	N	N	N	N	N	N	N	N	N	N	N	15.2	12	12	9
70	1140878	N	N	N	N	N	N	N	N	N	N	N	N	6.2	4.2	10.5	10.9
71	1143678	N	N	N	N	N	N	Y	N	N	N	N	N	7.6	14.8	13.5	13.2
72	1128230	N	N	N	Y	N	N	N	N	N	N	N	N	3.6	8.7	6.8	10.1
73	1120956	N	N	N	N	N	N	N	N	N	N	N	N	3.7	10.1	8.5	9.6
74	1121162	Y	N	N	N	N	N	N	Y	N	N	N	N	24.2	8.7	10.4	9.7
75	1122909	N	N	N	N	N	N	N	N	N	N	N	N	4.4	14.7	15	11.9
76	1112819	N	N	N	N	N	N	Y	N	N	N	N	N	8.2	11.7	13	14
77	1110990	N	N	N	N	N	N	Y	N	N	N	N	N	9.2	11.5	12.9	13.1
78	1101303	N	N	N	N	N	N	N	N	N	N	N	N	5.6	8.7	10.1	10.7
79	1090639	N	N	N	N	N	N	N	N	N	N	N	N	4.7	6	11.8	11.5
80	1086931	N	N	N	N	Y	N	N	N	N	N	N	N	9	6.4	12	9.2
81	1079522	N	N	N	N	N	N	N	N	Y	Y	N	N	6.4	7.4	9.7	9
82	1071064	Y	N	N	N	N	N	Y	Y	N	N	N	N	6.5	6.4	14.1	13
83	1030661	Y	Y	Y	N	N	N	N	Y	N	N	N	N	5.9	9.6	13.6	14
84	1308835	N	N	N	N	N	N	N	N	N	N	N	N	43.5	14	8.7	7.2
85	1295666	Y	N	N	N	N	N	N	Y	N	N	N	N	34.1	12.7	12.3	12
86	1311078	N	Y	N	N	N	N	Y	N	N	N	N	N	12.5	10	13.3	14
87	1313325	Y	N	N	N	N	N	Y	Y	N	N	N	N	1.4	0.6	10.8	6.3
88	1315554	N	N	N	N	N	Y	N	N	N	N	N	N	35	14	10.1	8.4
89	1106578	N	N	N	N	N	N	N	N	N	N	N	N	10.84	14	11.1	11
90	1318508	Y	N	N	N	N	Y	Y	Y	Y	N	N	N	2.6	7.5	9.5	10.5
91	1293775	N	N	N	N	N	N	N	N	N	N	N	N	3.1	8.5	9.4	9.9
92	1302433	Y	N	N	N	N	Y	N	Y	N	N	N	N	5.3	7.2	6.1	7.1
93	412900	Y	N	N	N	N	N	N	Y	N	N	N	N	22.4	13	10.8	8.5
94	1302993	N	N	Y	N	N	N	N	N	N	N	N	N	7.3	11	12.3	12.4
95	1270231	Y	N	Y	N	N	Y	Y	Y	N	N	N	N	4.9	0.9	11.4	8.6
96	1303493	N	N	N	N	N	N	Y	N	N	N	N	N	8.4	7.4	13.2	12.6
97	1296993	N	Y	N	N	N	Y	Y	N	N	Y	N	N	65	32	9.4	9.6
98	1295978	N	N	N	N	N	Y	N	N	N	N	N	N	5.1	7.4	8.1	11.8
99	1305231	N	N	Y	N	N	N	Y	N	N	N	N	N	22.4	17.2	12	12.2
100	1306683	N	N	N	N	N	Y	Y	N	N	N	N	N	9.5	-	14	-
101	1305964	N	N	N	N	N	N	N	N	N	N	N	N	5.2	7.4	15	8.2
102	1307311	N	N	N	N	N	N	Y	N	N	N	N	N	46.2	14	3.6	10.2
103	1306643	N	N	N	N	N	N	N	N	N	N	N	N	4	5.6	11.6	10.8
104	1316162	N	N	N	N	N	N	Y	N	N	N	N	N	7.9	3.4	10.5	8
105	1245032	Y	N	N	N	N	N	Y	Y	N	N	N	N	3.2	4.9	8.4	9.2
106	1318459	N	N	N	N	N	N	N	N	N	N	N	N	13.1	13	5.6	8.2
107	1318429	N	Y	N	N	N	Y	N	N	N	Y	N	N	11.2	11	7	6.1
108	1399640	N	N	N	N	N	N	Y	N	Y	Y	N	N	5.3	4.5	14.7	13.5

Sr. No	MRD NO	PLATELETS															LFT								RFT		
		PLT (1)	PLT (2)	PLT (3)	PLT (4)	PLT (5)	PLT (6)	PLT(7)	PLT(8)	PLT(9)	PLT(10)	PLT(15)	PLT(20)	PLT (DISCHARGE/ DAMA/	DAY AT WHICH PLT >30K	PLT(F/U)	BIL (t)	BIL(d)	SGOT	SGPT	ALP	ALBUMIN	TOTAL PROTEINS	UREA	CREATININE	Na
55	1203614	19	70	72	97	127	-	-	-	-	-	-	-	127	DAY2	135	1.65	0.55	450	320	188	3.9	6.2	29	0.92	137
56	1203172	4	6	7	15	22	50	62	79	73	142	177	-	177	DAY6	183			41	42				33	0.63	139
57	1204193	10	4	6	11	18	26	11	20	22	-	-	-	22	-	26	1.01	0.35	23	31	57	3.4	5.3	35	0.79	137
58	119338	12	26	18	27	91	58	-	-	-	-	-	-	58	DAY5	96	0.48	0.19	97	60				21	0.8	134
59	1193870	14	38	45	30	20	40	44	66	-	-	-	-	66	DAY2	72	2.06	1.55	227	118	1030	3.8	7.4	58	1.83	131
60	1196407	18	25	35	61	-	-	-	-	-	-	-	-	61	DAY3	118			280	173		4.15	6.4	34	0.67	124
61	1042338	15	10	20	24	24	52	118	116	106	100	102	-	110	DAY6	-	0.45	0.19	29	24	73	3.45	6.1	21	0.65	145
62	1138743	10	30	34	40	50	60	-	-	-	-	-	-	60	DAY2	78	0.46	0.07	32	18	81	4.42	7.7	76	0.68	138
63	1138761	6	24	22	21	60	150	-	-	-	-	-	-	150	DAY5	164	1.53	0.58	116	102	211	2.4	5.4	173	2.2	125
64	1135959	15	10	20	14	25	40	70	124	-	-	-	-	124	DAY6	132	0.8	0.25	40	78	53	3.9	6.8	39	0.66	139
65	1018578	10	6	6	6	8	10	8	-	-	-	-	-	8	-	9	1.6	0.78	34	19	125	2.92	6.7	45	1.05	143
66	260600	19	22	60	40	40	43	40	40	68	-	-	-	68	DAY3	72	0.62	0.21	26	81	61	4.01	6.4	32	0.85	139
67	432189	19	22	24	11	10	18	-	-	-	-	-	-	18	-	-	1.3	0.82	20	20	97	3.5	6	152	2.54	128
68	1138743	10	30	34	40	50	62	-	-	-	-	-	-	62	DAY2	77	0.46	0.07	32	18	81	4.4	7.7	36	0.68	138
69	1138761	6	18	22	26	80	150	164	-	-	-	-	-	164	DAY5	171	1.53	0.58	116	102	211	2.42	5.4	173	2.2	125
70	1140878	19	40	20	30	30	36	36	30	22	-	-	-	22	-	25			71	39				54	1.11	133
71	1143678	6	14	34	24	80	104	-	-	-	-	-	-	104	DAY3	123	0.52	0.15	33	45	89	4.33	6.9	19	0.84	139
72	1128230	10	10	4	5	6	13	16	33	74	79	94	-	94	DAY8	98	0.61	0.17	55	44	68	4	7.9	19	0.57	136
73	1120956	10	20	30	22	33	43	50	108	-	-	-	-	108	DAY3	111	0.41	0.22	47	29	102	3.01	5.6	38	1.27	138
74	1121162	13	26	25	37	42	44	43	48	49	53	154	201	352	DAY4	444	4.25	3.41	1128	840	23	2.35	4	83	4.76	139
75	1122909	19	38	70	114	183	239	-	-	-	-	-	-	239	DAY2	256	0.52	0.11	213	165	149	3.7	6.6	26	0.64	132
76	1112819	4	8	28	46	72	104	-	-	-	-	-	-	104	DAY4	109	1.34	0.38	25	37	109	3.7	6.6	11	0.82	144
77	1110990	4	10	10	10	12	10	8	50	-	-	-	-	50	DAY8	56	0.66	0.27	30	26	147	4.86	7.4	15	0.7	139
78	1101303	6	34	24	22	32	42	48	60	-	-	-	-	60	DAY2	68	0.21	0.14	43	50	100	4.24	7	27	0.57	142
79	1090639	4	8	6	16	39	40	60	-	-	-	-	-	60	DAY5	-	1.11	0.47	26	35	95	3.89	6.3	24	1.18	132
80	1086931	10	14	18	16	22	24	46	67	79	91	97	96	102	DAY7	110	1.35	0.53	70	45	224	2.64	5.2	157	4.58	119
81	1079522	10	14	18	34	31	50	150	-	-	-	-	-	150	DAY4	344	21	18.6	204	155	556	2.5	4.9	29	0.8	131
82	1071064	8	8	15	30	35	36	49	58	61	96	-	-	118	DAY4	201	0.3	0.13	32	61	92	4.3	7.3	16	0.51	141
83	1030661	6	12	18	34	42	44	65	98	-	-	-	-	98	DAY4	110	1.68	1.17	354	445	123			14	0.75	138
84	1308835	14	18	18	22	29	44	68	156	-	-	-	-	156	DAY6	188	2.94	2.32	77	125	236	2.5	5.4	151	6.28	139
85	1295666	6	8	8	8	10	12	12	15	22	27	29	-	29	-	29	0.71	0.26	47	41	158	1.9	4	80	2.85	132
86	1311078	4	32	70	67	80	121	-	-	-	-	-	-	121	DAY2	140	0.64	0.13	22	23	83	4.5	7.7	40	1.05	138
87	1313325	19	26	35	44	48	55	-	-	-	-	-	-	54	DAY3	56	0.86	0.25	29	8	107	2.5	5.7	290	3.01	151
88	1315554	19	34	24	22	18	30	40	40	60	80	629	-	497	DAY2	501	7.39	6.21	841	165	73	3.6	5.8	90	4.75	141
89	1106578	16	9	17	24	37	48	70	80	90	-	-	-	90	DAY5	98	0.5	0.08	18	11	70	3.9	0.9	203	8.6	139
90	1318508	12	54	70	81	79	81	82	82	87	90	90	92	110	DAY2	123	2.38	1.5	82	70	130	3.5	6.8	82	3.6	140
91	1293775	17	14	42	30	98	100	116	110	-	-	-	-	110	DAY3	154	3.07	1.23	96	42	85	3.4	6	31	0.68	133
92	1302433	8	18	31	35	42	54	71	78	84	87	-	-	98	DAY3	102	4.23	2.5	110	31	73	1.7	5.8	142	2.52	136
93	412900	10	41	22	22	80	54	77	121	155	162	-	-	155	DAY5	155	6.64	5.53	124	74	490	2.9	5.2	84	1.26	108
94	1302993	8	30	40	88	-	-	-	-	-	-	-	-	88	DAY3	102	0.64	0.15	54	98	79	4.4	7.8	23	0.58	142
95	1270231	14	10	28	-	-	-	-	-	-	-	-	-	28	-	-	3.42	3.1	105	76	2662	1.4	3.1	181	1.5	130
96	1303493	12	25	40	46	88	154	-	-	-	-	-	-	154	DAY3	163	2.43	1.04	299	139	178	2.8	5.2	90	1.48	134
97	1296993	19	16	-	-	-	-	-	-	-	-	-	-	16	-	-	6.83	3.86	45	36	83	2.6	5.5	150	2.83	146
98	1295978	19	24	20	26	40	28	30	-	-	-	-	-	30	DAY7	52	0.39	0.3	29	12	7.5	2.3	4.8	60	1.15	126
99	1305231	19	26	24	60	71	128	-	-	-	-	-	-	128	DAY4	132	0.64	0.3	16	29	46	3.7	6.2	32	0.63	143
100	1306683	12	-	-	-	-	-	-	-	-	-	-	-	12	-	-	8.72	6.09	820	519	71	3.09	5.6	102	4.78	139
101	1305964	19	24	12	10	42	24	24	-	-	-	-	-	24	DAY12	42	6.11	4.8	1049	478	194	3.8	7.2	84	4.21	138
102	1307311	18	10	40	42	24	52	86	92	157	211	-	-	252	DAY3	255	4.75	3.62	850	404	236	2.22	4.3	116	4.13	145
103	1306643	15	70	48	80	40	52	55	76	78	-	-	-	78	DAY2	98	1.72	0.92	21	36	43	2.2	3.8	45	0.27	152
104	1316162	19	40	24	14	-	-	-	-	-	-	-	-	14	-	-	6.76	4.36	589	294	83	2.7	4.8	91	4.5	140
105	1245032	17	20	16	54	52	-	-	-	-	-	-	-	52	DAY4	74	0.28	0.05	117	37	171	2.34	6.1	29	0.9	140
106	1318459	12	80	83	98	84	62	67	74	79	98	-	-	98	DAY2	101	0.45	0.37	32	21	251	1.2	4	155	5	136
107	1318429	19	14	-	-	-	-	-	-	-	-	-	-	14	-	-	17	12	77	33	30	2.39	5	45	2.56	145
108	1399640	16	13	27	44	96	-	-	-	-	-	-	-	96	DAY4	102	0.31	0.19	133	80	79	3.4	6.5	19	0.67	139

Sr. No	MRD NO				BONE MARROW				AUTOIMMUNE PROFILE		VIRAL MARKERS
		K	Cl	PBF	IF DONE	CELLULARITY	MEGAKARYOCYTES	OTHER	ANA	APLA	
55	1203614	3.97	97	NCNC	N	-	-	-	-	-	NR
56	1203172	3.45	100	NCNC	Y	HYPER	-	ITP	ANAIF 3+,ANA PROFILE -	POSITIVE FOR LUPUS ANTICOAGULANT	NR
57	1204193	3.58	103	MACROCYTES WITH HYPERSEGMENTED NEUTROPHILS	Y	HYPER	Y	ITP	-	-	NR
58	119338	5.49	100	NCNC	N	-	-	-	-	-	NR
59	1193870	7.1	100	NCNC	N	-	-	-	-	-	NR
60	1196407	5	89	NCNC	N	-	-	-	-	-	NR
61	1042338	3.2	103	NCNC	N	-	-	-	-	-	NR
62	1138743	3.21	106	NCNC	Y	NORMO	Y	ITP	ANA IF 1+ ANA PROFILE -	-	NR
63	1138761	4.6	98	NCNC,TOXIC GRANULES PRESENT	N	-	-	-	-	-	NR
64	1135959	3.8	99	NCNC	Y	HYPER	Y	ITP	-	-	NR
65	1018578	4	111	NCNC	Y	HYPER	Y	ITP	ANAIF -	-	NR
66	260600	4.69	103	NCNC	y	HYPER	Y	ITP	-	-	NR
67	432189	5.16	93	MCHC	Y	HYPER	N	ERYTHROID PROMINENCE	ANA IF 2+ ANCA -	-	NR
68	1138743	3.21	106	NCNC	Y	HYPER	Y	ITP	ANA IF 1+ ANA PROFILE -	-	NR
69	1138761	4.66	98	NCNC ,TOXIC GRANULES PRESENT	N	-	-	-	-	-	NR
70	1140878	3.79	98	NCNC	Y	NORMO	N	GRANULOMATOUS INFLAMMATION	-	-	NR
71	1143678	3.68	101	NCNC	Y	NORMO	Y	ITP	ANA IF 2+	-	NR
72	1128230	3.67	107	NCNC	Y	HYPER	Y	ITP	ANA IF 3+ SSA /RO52+	-	NR
73	1120956	3.98	106	NCNC	N	-	-	-	-	-	NR
74	1121162	4.5	108	NCNC ,TOXIC GRANULES PRESENT	N	-	-	-	-	-	NR
75	1122909	4.39	101	NCNC	N	-	-	-	-	-	NR
76	1112819	3.82	105	NCNC	Y	NORMO	Y	ITP	-	-	NR
77	1110990	4.28	102	NCNC	Y	NORMO	Y	ITP	-	-	NR
78	1101303	3.82	107	NCNC	Y	HYPER	Y	ITP	-	-	NR
79	1090639	4.21	99	NCNC	Y	HYPER	N	ERYTHROID PROMINENCE	-	-	NR
80	1086931	6.41	91	NCNC,TOXIC GRANULES PRESENT	N	-	-	-	-	-	NR
81	1079522	4.14	105	NCNC	N	-	-	-	ANA IF 1+	-	NR
82	1071064	4.49	109	NCNC	Y	HYPER	Y	ITP	ANA IF 1+ AMAM2 +	-	NR
83	1030661	4.12	104	NCNC	Y	HYPER	Y	ITP	NEG	-	NR
84	1308835	4.01	104	NCNC	N	-	-	-	NEG	-	NR
85	1295666	3.58	102	NCNC	Y	NORMO	N	NORMAL	NEG	-	NR
86	1311078	4.47	101	NCNC	Y	NORMO	Y	ITP	ANA IF 1+	-	NR
87	1313325	4.87	119	NCNC	Y	HYPER	N	HEMOPHAGOCYTOSIS	-	-	NR
88	1315554	4.2	104	NCNC	N	-	-	-	ANA IF 1+	-	NR
89	1106578	4.7	110	MCHC	Y	HYPER	Y	ITP	NEG	-	NR
90	1318508	3.5	105	NCNC	N	-	-	-	NEG	-	NR
91	1293775	4.68	106	MACROCYTES SEEN	N	-	-	-	-	-	NR
92	1302433	5.21	98	ANISOCYTOSIS,MACROCYTES,NC	N	-	-	-	-	-	NR
93	412900	4.9	74	MHHC	N	-	-	-	NEG	-	NR
94	1302993	4.09	105	NCNC	N	-	-	-	NEG	-	NR
95	1270231	3.7	94	NCNC ,TOXIC GRANULES PRESENT	N	-	-	-	ANA IF 3+ SSA /RO52+	-	NR
96	1303493	3.79	101	NCNC	N	-	-	-	-	-	NR
97	1296993	4.09	105	NCNC ,TOXIC GRANULES PRESENT	N	-	-	-	-	-	NR
98	1295978	4.24	97	ANISOCYTOSIS,MCHC	N	-	-	-	-	-	HEP C
99	1305231	3.5	107	NCNC	Y	NORMO	Y	ITP	NEG	-	NR
100	1306683	4.21	103	NCNC	N	-	-	-	-	-	HEP A
101	1305964	4.26	105	NCNC	N	-	-	-	NEG	-	NR
102	1307311	7.23	104	NCNC	N	-	-	-	NEG	-	NR
103	1306643	3.18	121	MCHC	N	-	-	-	-	-	NR
104	1316162	3.98	112	ANISOCYTOSIS,MCHC	N	-	-	-	NEG	-	NR
105	1245032	4.44	111	NCNC	Y	NORMO	Y	ITP WITH IDA	ANA IF 3+ SSA /RO52+,DSDNA+	-	NR
106	1318459	4.29	100	NCNC	N	-	-	-	-	-	NR
107	1318429	4.47	108	ANISOCYTOSIS,MACROCYTES,NC	N	-	-	-	-	-	NR
108	1399640	3.8	101	NCNC	N	-	-	-	NEG	-	NR

Sr. No	MRD NO	TROPICAL FEVER	D DIMER	PROTHROMBIN TIME	FIBRINOGEN	RADIOLOGICAL INVESTIGATION
						USG WHOLE ABDOMEN
55	1203614	DENGUE NS1	-	12	-	FATTY LIVER GRADE 1,MILD PLEURAL EFFUSION,MODERATE ASCITES
56	1203172	NEGATIVE	-	12.2	-	NAD
57	1204193	NEGATIVE	-	11.9	-	FATTY LIVER GRADE 1
58	119338	DENGUE NS1	-	11.9	-	-
59	1193870	LEPTOSPIRA IGM +	-	11.5	-	FATTY LIVER GRADE 1,REACTIVE GALL BLADDER WALL EDEMA
60	1196407	DENGUE NS1	-	10.3	-	GB WALL EDEMA WITH MINIMAL ASCITES AND PLEURAL EFFUSION
61	1042338	DENGUE IGM	-	11.4	-	UNCOMPLIATED CHOLELITHIASIS
62	1138743	DENGUE NS1	-	11.1	-	-
63	1138761	NEGATIVE	-	11.5	-	HEPATOMEGALY WITH FATTY LIVER GRADE 1
64	1135959	NEGATIVE	-	10.4	-	NAD
65	1018578	LEPTOSPIRA IGM +	-	13.6	-	CHRONIC LIVER DISEASE
66	260600	NEGATIVE	-	11.7	-	NAD
67	432189	NEGATIVE		16.2	-	CLD WITH PORTAL HYPERTENSION
68	1138743	DENGUE IGM	-	11.1	-	MILD SPLENOMEGALY
69	1138761	NEGATIVE	-	11.2	-	HEPATOMEGALY WITH FATTY LIVER GRADE 1
70	1140878	NEGATIVE	-	11.3	-	NAD
71	1143678	NEGATIVE	-	11.2	-	FATTY LIVER GRADE 1
72	1128230	NEGATIVE	-	11.3	-	FATTY LIVER GRADE 1
73	1120956	DENGUE IGM ,SCHIZONT AND TROPHOZOITE FORMS OF PLASMODIUM VIVAX SEEN	-	13.6	-	SPLENOMEGALY
74	1121162	NEGATIVE	-	19.5	-	MISSED ABORTION WITH MILD ASCITES
75	1122909	DENGUE IGM	-	11.1	-	FATTY LIVER GRADE 1
76	1112819	NEGATIVE	-	12.6	-	FATTY LIVER GRADE 1
77	1110990	NEGATIVE	-	14.7	-	NAD
78	1101303	NEGATIVE	-	12.1	-	UNCOMPLIATED CHOLELITHIASIS
79	1090639	LEPTOSPIRA IGM +	-	11.9	-	NAD
80	1086931	NEGATIVE	-	11.3	-	LEFT PYELITIS
81	1079522	LEPTOSPIRA IGM +	-	14.4	-	HEPATOSPLENOMEGALY
82	1071064	NEGATIVE	-	11.7	-	FATTY LIVER GRADE 1
83	1030661	NEGATIVE	346	12.3	-	MILD SPLENOMEGALY
84	1308835	NEGATIVE	33.22	11.1	398	RENAL PARENCHYMAL CHANGES GRADE 1
85	1295666	NEGATIVE	-	26.4	-	POST RIGHT NEPHRECTOMY ,INFECTIVE ETIOLOGY IN LEFT KIDNEY
86	1311078	NEGATIVE	-	12.2	-	NAD
87	1313325	NEGATIVE	-	13.3	-	MODERATE LEFT HYDROURETRONEPHROSIS
88	1315554	NEGATIVE	-	20.2	-	GB SLUDGE,MILD ASCITES,MILG RIGHT PLEURAL EFFUSION
89	1106578	NEGATIVE	-	13.7	-	CLD,CHOLELITHIASIS
90	1318508	NEGATIVE	-	11.4	-	HEPATOMEGALY WITH FATTY LIVER GRADE 1
91	1293775	NEGATIVE	-	19.8	-	FATTY LIVER GRADE 1
92	1302433	NEGATIVE	-	28.5	-	RIGHT NEPHROLITHIASIS WITH HUN
93	412900	NEGATIVE	-	16.9	-	ACUTE ON CHRONIC PANCREATITIS,GB WALL EDEMA
94	1302993	NEGATIVE	-	15	-	NAD
95	1270231	NEGATIVE	-	16.6	181	RENAL PARENCHYMAL CHANGES GRADE 1
96	1303493	NEGATIVE	-	12	-	NAD
97	1296993	NEGATIVE	-	37.4	-	CIRRHOSSIS WITH PORTAL HYPERTENSION
98	1295978	NEGATIVE	-	11.8	-	RENAL PARENCHYMAL CHANGES GRADE 1
99	1305231	NEGATIVE	-	10.4	-	NAD
100	1306683	NEGATIVE	-	27.8	-	REACTIVE GB WALL EDEMA
101	1305964	NEGATIVE	-	22.5	-	NAD
102	1307311	NEGATIVE	4.2	14.1	-	NAD
103	1306643	NEGATIVE	-	11.4	-	NAD
104	1316162	NEGATIVE	-	0	-	REACTIVE GB WALL EDEMA
105	1245032	NEGATIVE	-	11.4	-	FATTY LIVER GRADE 1
106	1318459	NEGATIVE	-	17.2	-	RENAL PARENCHYMAL CHANGES GRADE 1
107	1318429	NEGATIVE	-	75.8	-	CIRRHOSSIS WITH PORTAL HYPERTENSION
108	1399640	NEGATIVE	-	9.8	-	HEPATOSPLENOMEGALY

Sr. No	MRD NO		RDP	SDP	RDP + SDP	INTERVENTION								
		OTHER CT				RDP	SDP	STEROIDS			TPO AGONIST		
								DRUG	DOSE	DURATION	DRUG USED	DOSE	DURATION
55	1203614	-	N	Y	N	-	1	-	-	-	-	-	-
56	1203172	-	N	Y	N	-	4	METHYLPREDNISOLONE	1G	3D	-	-	-
57	1204193	-	N	Y	N	-	4	PREDNISOLONE	30MG	5D	-	-	-
58	119338	-	N	N	N	-	-	-	-	-	-	-	-
59	1193870	-	N	Y	N	-	2	-	-	-	-	-	-
60	1196407	-	N	Y	N	-	1	-	-	-	-	-	-
61	1042338	-	Y	Y	Y	4	1	-	-	-	-	-	-
62	1138743	-	N	N	N	-	-	METHYLPREDNISOLONE	1G	3D	-	-	-
63	1138761	-	N	N	N	-	-	-	-	-	-	-	-
64	1135959	-	N	N	N	-	-	METHYLPREDNISOLONE	1G	3D	-	-	-
65	1018578	-	Y	Y	Y	4	1	-	-	-	-	-	-
66	260600	-	N	N	N	-	-	PREDNISOLONE	60MG	3D	-	-	-
67	432189	-	N	Y	N	-	1	-	-	-	-	-	-
68	1138743	-	N	Y	N	-	1	-	-	-	-	-	-
69	1138761	-	Y	Y	Y	4	1	-	-	-	-	-	-
70	1140878	-	N	Y	N	-	1	-	-	-	-	-	-
71	1143678	-	N	Y	N	-	1	METHYLPREDNISOLONE	500MG	3D	-	-	-
72	1128230	-	Y	Y	Y	9	1	DEXAMETHASONE	8MG	5D	-	-	-
73	1120956	-	N	Y	N	-	1	-	-	-	-	-	-
74	1121162	-	Y	N	N	4	-	-	-	-	-	-	-
75	1122909	-	Y	N	N	4	-	-	-	-	-	-	-
76	1112819	-	N	N	N	-	-	PREDNISOLONE	60MG	3D	-	-	-
77	1110990	-	Y	Y	Y	2	4	DEXAMETHASONE	8MG	3D	-	-	-
78	1101303	-	Y	N	N	4	-	DEXAMETHASONE	8MG	3D	-	-	-
79	1090639	-	N	Y	N	-	1	-	-	-	-	-	-
80	1086931	-	Y	Y	Y	2	2	-	-	-	-	-	-
81	1079522	-	Y	N	N	2	-	-	-	-	-	-	
82	1071064	-	N	Y	N	-	1	DEXAMETHASONE	8MG	3D	-	-	-
83	1030661	-	Y	N	N	4	-	METHYLPREDNISOLONE	1G	3D	-	-	-
84	1308835	-	Y	N	N	4	-	HYDROCORTISONE	150MG	8D	-	-	-
85	1295666	-	N	Y	N	-	1	PREDNISOLONE	60MG	3D	-	-	-
86	1311078	-	Y	Y	Y	4	1	METHYLPREDNISOLONE	1G	3D	-	-	-
87	1313325	-	Y	N	N	2	-	-	-	-	-	-	-
88	1315554	-	N	N	N	-	-	HYDROCORTISONE	150MG	10D	-	-	-
89	1106578	-	Y	N	N	2	-	PREDNISOLONE	60MG	3D	-	-	-
90	1318508	-	N	Y	N	-	1	-	-	-	-	-	-
91	1293775	-	Y	N	N	2	-	-	-	-	-	-	-
92	1302433	-	N	Y	N	-	1	-	-	-	-	-	-
93	412900	-	N	Y	N	-	1	-	-	-	-	-	-
94	1302993	-	N	Y	N	-	1	-	-	-	-	-	-
95	1270231	-	N	Y	N	-	1	-	-	-	-	-	-
96	1303493	-	N	Y	N	-	1	-	-	-	-	-	-
97	1296993	-	N	Y	N	-	2	-	-	-	-	-	-
98	1295978	-	N	N	N	-	-	-	-	-	-	-	-
99	1305231	-	N	N	N	-	-	DEXAMETHASONE	8MG	3D	-	-	-
100	1306683	-	Y	N	N	2	-	HYDROCORTISONE	150MG	2D	-	-	-
101	1305964	-	Y	N	N	2	-	-	-	-	-	-	-
102	1307311	-	N	Y	N	-	2	-	-	-	-	-	-
103	1306643	-	Y	N	N	2	-	-	-	-	-	-	-
104	1316162	-	Y	N	N	4	-	HYDROCORTISONE	150MG	3D	-	-	-
105	1245032	-	Y	N	N	4	-	METHYLPREDNISOLONE	1000MG	3D	-	-	-
106	1318459	-	N	Y	N	-	1	HYDROCORTISONE	300MG	10D	-	-	-
107	1318429	-	N	Y	N	-	2	-	-	-	-	-	-
108	1399640	-	N	Y	N	-	1	-	-	-	-	-	-

Sr. No	MRD NO	OUTCOMES								
		OTHER INTERVENTION (IVIG/SPLENECTOMY	COMPLETE	PARTIAL	NO RESPONSE	LENGTH OF HOSPITAL STAY	NEED FOR ICU CARE	NEED FOR		DISCHARGE /DAMA/ MORTALITY
								Y/N	DURATION	
55	1203614	-	Y	-	-	5D	N	N	-	DISCHARGE
56	1203172	-	Y	-	-	11D	Y	Y	6D	DAMA
57	1204193	-	-	-	Y	9D	Y	N	-	DISCHARGE
58	119338	-	Y	-	-	6D	N	N	-	DISCHARGE
59	1193870	-	-	Y	-	8D	Y	N	-	DISCHARGE
60	1196407	-	Y	-	-	4D	N	N	-	DISCHARGE
61	1042338	-	Y	-	-	19D	Y	N	-	DAMA
62	1138743	-	-	Y	-	6D	N	N	-	DISCHARGE
63	1138761	-	Y	-	-	6D	N	N	-	DISCHARGE
64	1135959	-	Y	-	-	8D	N	N	-	DISCHARGE
65	1018578	-	-	-	-	7D	N	N	-	DAMA
66	260600	-	-	Y	-	9D	N	N	-	DISCHARGE
67	432189	-	-	-	-	6D	Y	Y	5D	DAMA
68	1138743	-	-	Y	-	6D	N	N	-	DISCHARGE
69	1138761	-	Y	-	-	7D	N	N	-	DISCHARGE
70	1140878	-	-	-	y	10D	N	N	-	DISCHARGE
71	1143678	-	Y	-	-	6D	N	N	-	DISCHARGE
72	1128230	-	-	Y	-	11D	N	N	-	DISCHARGE
73	1120956	-	Y	-	-	8D	N	N	-	DISCHARGE
74	1121162	-	Y	-	-	38D	Y	Y	15D	DISCHARGE
75	1122909	-	Y	-	-	6D	N	N	-	DISCHARGE
76	1112819	-	Y	-	-	6D	N	N	-	DISCHARGE
77	1110990	-	-	Y	-	10D	N	N	-	DISCHARGE
78	1101303	-	-	Y	-	10D	N	N	-	DISCHARGE
79	1090639	-	-	Y	-	7D	N	N	-	DAMA
80	1086931	-	Y	-	-	34D	Y	Y	26D	DISCHARGE
81	1079522	-	Y	-	-	7D	Y	N	-	DISCHARGE
82	1071064	-	Y	-	-	12D	N	N	-	DISCHARGE
83	1030661	-	y	-	-	8D	N	N	-	DISCHARGE
84	1308835	-	Y	-	-	8D	Y	N	-	DISCHARGE
85	1295666	-	-	-	Y	13D	Y	N	-	DISCHARGE
86	1311078	-	Y	-	-	6D	N	N	-	DISCHARGE
87	1313325	-	-	Y	-	6D	Y	N	-	DAMA
88	1315554	-	Y	-	-	18D	Y	N	-	DAMA
89	1106578	-	-	Y	-	8D	Y	N	-	DISCHARGE
90	1318508	-	Y	-	-	21D	Y	Y	10D	DISCHARGE
91	1293775	-	Y	-	-	8D	N	N	-	DISCHARGE
92	1302433	-	Y	-	-	13D	N	N	-	DISCHARGE
93	412900	-	Y	-	-	13D	Y	Y	6D	DISCHARGE
94	1302993	-	Y	-	-	5D	N	N	-	DISCHARGE
95	1270231	-	-	-	Y	3D	Y	Y	2D	MORTALITY
96	1303493	-	Y	-	-	6D	N	N	-	DISCHARGE
97	1296993	-	-	-	-	2D	Y	N	-	DAMA
98	1295978	-	-	Y	-	7D	N	N	-	DISCHARGE
99	1305231	-	Y	-	-	6D	N	N	-	DISCHARGE
100	1306683	-	-	-	Y	2D	Y	Y	2D	DAMA
101	1305964	-	-	Y	-	7D	Y	Y	6D	DAMA
102	1307311	-	Y	-	-	11D	Y	Y	6D	DAMA
103	1306643	-	-	Y	-	9D	Y	Y	2D	DISCHARGE
104	1316162	-	-	-	Y	4D	Y	Y	3D	DAMA
105	1245032	-	-	Y	-	5D	Y	N	-	DISCHARGE
106	1318459	-	Y	-	-	10D	Y	N	-	DISCHARGE
107	1318429	-	-	-	Y	2D	Y	Y	2D	MORTALITY
108	1399640	-	Y	-	-	5D	N	N	-	DISCHARGE

Sr. No	MRD NO	INITIALS	AGE	SEX	DATE OF ADMISSION	ETIOLOGY	SUB - ETIOLOGY	ALCOHOLIC HISTORY				H/O TREATMENT (CHEMOTHERAPY, HEPARIN.ABCIXIMAB, QUININE,			COMORBIDITIES	
								YES/ NO	DURATION	AMOUNT	TYPE	YES /NO	DRUG	DURATION OF DRUG GIVEN	TYPE 2 DM	HYPERTENSION
109	1401892	GS	62	M	22-10-2021	ITP	-	N	-	-	-	N	-	-	Y	Y
110	1401943	KC	20	M	22-10-2021	TROPICAL FEVER	-	N	-	-	-	N	-	-	N	N
111	140969	MS	33	F	23-10-2021	TROPICAL FEVER	-	N	-	-	-	N	-	-	N	N
112	1402043	SD	66	F	23-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	Y	Y
113	1402045	JS	70	M	23-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
114	1403216	TS	72	M	26-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	Y	Y
115	1404307	KS	34	M	28-10-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
116	1406282	AA	42	F	01-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
117	1406907	RK	36	F	01-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
118	1406967	VS	55	M	01-11-2021	MULTIFACTORIAL	RESPIRATORY ,GENITOURINARY,SEPSIS,TROPICAL FEVER ,HLH	N	-	-	-	N	-	-	N	N
119	1407693	PKC	57	M	03-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
120	1350680	RK	45	M	03-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
121	1409007	AR	64	M	04-11-2021	TROPICAL FEVER	DENGUE,SCRUB	N	-	-	-	N	-	-	N	N
122	1409624	R	21	M	05-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N
123	1409765	NK	37	M	06-11-2021	TROPICAL FEVER	DENGUE	N	-	-	-	N	-	-	N	N

Sr. No	MRD NO								SYMPTOMS/SIGNS									
		BRONCHIAL ASTHMA	COAD	CAD	CVA	CKD	THYROID DISEASE	ANY OTHER	FEVER	MALAISE/FATIGUE/GENERALISED BODY WEAKNESS	NAUSEA	VOMITING	DIARRHOEA	CONSTIPATION	BREATHLESSNSS	BLEEDING MANIFESTATIONS		
																SUBCONJUNCTIVAL HEMORRHAGE	EPISTAXIS	GUM BLEEDING
109	1401892	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y
110	1401943	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
111	140969	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	N
112	1402043	N	N	N	N	N	N	N	Y	Y	Y	N	N	N	N	N	N	N
113	1402045	N	N	N	N	N	N	N	Y	Y	Y	N	N	N	N	N	N	N
114	1403216	N	N	N	N	N	N	N	Y	Y	Y	N	N	N	N	N	N	N
115	1404307	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
116	1406282	N	N	N	N	N	N	N	Y	Y	N	Y	N	N	N	N	N	N
117	1406907	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
118	1406967	N	N	N	N	N	N	N	Y	Y	N	N	N	N	Y	N	N	N
119	1407693	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
120	1350680	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
121	1409007	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
122	1409624	N	N	N	N	N	N	N	Y	Y	N	Y	Y	N	N	N	N	N
123	1409765	N	N	N	N	N	N	N	Y	N	Y	Y	N	N	N	N	N	N

Sr. No	MRD NO													LAB PARAMETERS			
														TLC (counts/μL)		Hb	
		ECHYMOSES	MALENA	HEMATURIA	MENORRHAGIA	WEIGHT LOSS	ABDOMINAL PAIN	RED SPOTS	RASHES	HEPATOMEGALY	SPLENOMEGALY	LYMPHADENOPATHY	OTHER	TLC (DOA)	TLC (F/U)	Hb (DOA)	Hb(F/U)
109	1401892	Y	N	N	N	N	N	N	Y	N	N	N	N	13.6	9.2	12	11.5
110	1401943	N	N	N	N	N	Y	N	N	N	N	N	N	2.5	5	16	14
111	140969	N	N	N	N	N	N	Y	N	N	N	N	N	8.7	8.6	14.6	14.3
112	1402043	N	N	N	N	N	Y	N	N	N	N	N	N	2.3	4.5	8.8	8.5
113	1402045	N	N	N	N	N	Y	N	N	N	N	N	N	6.4	7.2	15.1	13
114	1403216	N	N	N	N	N	N	N	N	N	N	N	N	4.2	6.4	15.7	13
115	1404307	N	N	N	N	N	N	N	N	N	N	N	N	4	5	13.6	13.8
116	1406282	N	N	N	Y	N	N	N	N	Y	Y	N	N	9.7	6.2	12.3	11
117	1406907	N	N	N	N	N	Y	N	N	N	N	N	N	4.1	5.4	15	14.9
118	1406967	N	N	N	N	N	N	N	N	Y	Y	N	N	5.4	6.3	9.7	6.3
119	1407693	N	Y	N	N	N	N	N	N	N	Y	N	N	4.2	4.5	15	12.9
120	1350680	Y	N	N	N	N	N	N	Y	N	N	N	N	4.8	6.5	17	14
121	1409007	N	N	N	N	N	Y	N	N	N	N	N	N	2.2	5	13.3	11.5
122	1409624	N	Y	N	N	N	N	N	N	N	N	N	N	2.9	4.2	16.5	14.1
123	1409765	N	N	N	N	N	Y	N	N	Y	Y	N	N	4.8	7.2	16.3	14

Sr. No	MRD NO	PLATELETS															LFT								RFT		
		PLT (1)	PLT (2)	PLT (3)	PLT (4)	PLT (5)	PLT (6)	PLT(7)	PLT(8)	PLT(9)	PLT(10)	PLT(15)	PLT(20)	PLT (DISCHARGE/ DAMA/	DAY AT WHICH PLT >30K	PLT(F/U)	BIL (t)	BIL(d)	SGOT	SGPT	ALP	ALBUMIN	TOTAL PROTEINS	UREA	CREATININE	Na
109	1401892	6	9	13	28	31	45	54	67	78	82	88	98	110	DAY5	140	0.58	0.21	17	29	48	4.7	7.1	40	1.09	132
110	1401943	19	24	44	64	60	110	-	-	-	-	-	-	110	DAY3	156	0.64	0.2	57	43	60	3.5	6.5	45	1.1	130
111	140969	18	23	45	48	51	57	-	-	-	-	-	-	57	DAY3	78	0.47	0.32	657	235	211	2.8	5.2	38	1.33	131
112	1402043	16	14	68	58	96	128	-	-	-	-	-	-	128	DAY3	143	0.92	0.16	100	35	116	3.4	6.2	14	0.61	131
113	1402045	14	24	30	52	130	124	-	-	-	-	-	-	124	DAY3	136	0.62	0.16	177	134	80	3.7	6.1	38	1.33	139
114	1403216	12	28	40	26	46	-	-	-	-	-	-	-	46	DAY9	78	0.56	0.22	145	98	147	3.3	5.8	37	1.19	130
115	1404307	12	38	66	-	-	-	-	-	-	-	-	-	66	DAY2	76	0.37	0.21	266	104	108	3.25	5.2	12	0.65	143
116	1406282	12	18	30	50	-	-	-	-	-	-	-	-	50	DAY3	92	0.83	0.61	213	192	282	3.06	5.8	39	1.02	138
117	1406907	15	49	63	72	144	-	-	-	-	-	-	-	144	DAY2	151	0.69	0.18	496	291	121	4.05	6.8	17	0.86	133
118	1406967	8	10	12	10	4	22	20	8	6	4	4	-	4	-	14	1.14	1.2	270	108	95	2.9	6.1	225	3.06	141
119	1407693	12	21	14	48	60	50	56	110	-	-	-	-	110	DAY4	150	1.32	0.61	1287	105	169	3.8	5.8	22	1.28	136
120	1350680	6	18	46	68	154	-	-	-	-	-	-	-	154	DAY3	159	0.84	0.65	81	71	298	3.9	6.5	23	0.76	134
121	1409007	18	16	18	36	64	96	-	-	-	-	-	-	96	DAY4	130	0.44	0.17	190	82	94	4.1	6.8	32	1.1	130
122	1409624	18	40	140	166	199	106	-	-	-	-	-	-	106	DAY3	123	1.08	0.64	138	42	98	3.5	5.9	36	1.09	132
123	1409765	10	44	68	42	62	72	199	-	-	-	-	-	199	DAY2	240	0.89	0.33	156	60	119	3.4	5.8	37	1.1	135

Sr. No	MRD NO				BONE MARROW				AUTOIMMUNE PROFILE		VIRAL MARKERS
		K	Cl	PBF	IF DONE	CELLULARITY	MEGAKARYOCYTES	OTHER	ANA	APLA	
109	1401892	4.35	101	NCNC	Y	NORMO	Y	ITP	NEG	-	NR
110	1401943	4.34	102	NCNC	N	-	-	-	NEG	-	NR
111	140969	5.7	103	NCNC	N	-	-	-	NEG	-	NR
112	1402043	4.44	97	NCNC	N	-	-	-	NEG	-	NR
113	1402045	4.08	103	NCNC	N	-	-	-	-	-	NR
114	1403216	5.31	99	NCNC	N	-	-	-	-	-	NR
115	1404307	3.72	108	NC,MACROCYTES	N	-	-	-	-	-	NR
116	1406282	4	104	NCNC	N	-	-	-	-	-	NR
117	1406907	4.17	100	NCNC	N	-	-	-	-	-	NR
118	1406967	5.5	101	NCNC	Y	NORMO	N	HEMOPHAGOCYTOSIS	-	-	NR
119	1407693	4.18	104	NCNC	N	-	-	-	-	-	NR
120	1350680	4.82	100	NCNC	N	-	-	-	-	-	NR
121	1409007	5.16	97	NCNC	N	-	-	-	-	-	NR
122	1409624	4.08	100	NCNC	N	-	-	-	-	-	NR
123	1409765	4.32	98	NCNC	N	-	-	-	-	-	NR

Sr. No	MRD NO	TROPICAL FEVER	D DIMER	PROTHROMBIN TIME	FIBRINOGEN	RADIOLOGICAL INVESTIGATION
						USG WHOLE ABDOMEN
109	1401892	NEGATIVE	-	12.1	-	FATTY LIVER GRADE 2
110	1401943	NEGATIVE	-	11.4	-	REACTIVE GB WALL EDEMA
111	140969	NEGATIVE	-	12.6	-	REACTIVE GB WALL EDEMA
112	1402043	DENGUE NS1	-	9.9	-	REACTIVE GB WALL EDEMA
113	1402045	DENGUE IGM	-	11	-	FATTY LIVER GRADE 1
114	1403216	DENGUE NS1	-	12.8	-	FATTY LIVER GRADE 1
115	1404307	DENGUE NS1	-	10.5	-	REACTIVE GB WALL EDEMA
116	1406282	DENGUE IGM	-	12	-	HEPATOSPLENOMEGALY
117	1406907	DENGUE NS1	-	10.7	-	REACTIVE GB WALL EDEMA
118	1406967	DENGUE NS1	-	14.7		HEPATOSPLENOMEGALY
119	1407693	DENGUE NS1	-	10.8	-	SPLENOMEGALY
120	1350680	DENGUE NS1	-	10.5	-	FATTY LIVER GRADE 1,MILD PLEURAL EFFUSION,MODERATE ASCITES
121	1409007	DENGUE IGM,SCRUB IGM	-	11.6	-	FATTY LIVER GRADE 1
122	1409624	DENGUE NS1	-	11.7	-	FATTY LIVER GRADE 1
123	1409765	DENGUE NS1	-	11.2	-	REACTIVE GB WALL EDEMA, HEPATOSPLENOMEGALY

Sr. No	MRD NO	OTHER CT	RDP	SDP	RDP + SDP	INTERVENTION							
						RDP	SDP	STEROIDS			TPO AGONIST		
								DRUG	DOSE	DURATION	DRUG USED	DOSE	DURATION
109	1401892	-	N	N	N	-	-	DEXAMETHASONE	8MG	3D	ROMIPLOSTIM	250MCG	5D
110	1401943	-	Y	N	N	2	-	-	-	-	-	-	-
111	140969	-	Y	Y	Y	2	1	-	-	-	-	-	-
112	1402043	-	Y	N	N	4	-	-	-	-	-	-	-
113	1402045	-	N	Y	N	-	1	-	-	-	-	-	-
114	1403216	-	Y	N	N	4	-	-	-	-	-	-	-
115	1404307	-	Y	Y	Y	2	1	-	-	-	-	-	-
116	1406282	-	N	Y	N	-	1	-	-	-	-	-	-
117	1406907	-	Y	N	N	4	-	-	-	-	-	-	-
118	1406967	-	Y	Y	Y	4	1	DEXAMETHASONE	8MG	3D	-	-	-
119	1407693	-	N	Y	N	-	1	-	-	-	-	-	-
120	1350680	-	Y	N	N	2	-	-	-	-	-	-	-
121	1409007	-	N	Y	N	-	1	-	-	-	-	-	-
122	1409624	-	N	Y	N	-	1	-	-	-	-	-	-
123	1409765	-	Y	N	N	2	-	-	-	-	-	-	-

Sr. No	MRD NO					OUTCOMES				
		OTHER INTERVENTION (IVIG/SPLENECTOMY	COMPLETE	PARTIAL	NO RESPONSE	LENGTH OF HOSPITAL STAY	NEED FOR ICU CARE	NEED FOR		DISCHARGE /DAMA/ MORTALITY
								Y/N	DURATION	
109	1401892	IVIG	Y	-	-	31D	Y	N	-	DISCHARGE
110	1401943	-	Y	-	-	6D	N	N	-	DISCHARGE
111	140969	-	-	Y	-	5D	N	N	-	DISCHARGE
112	1402043	-	Y	-	-	5D	N	N	-	DISCHARGE
113	1402045	-	Y	-	-	6D	N	N	-	DISCHARGE
114	1403216	-	-	Y	-	5D	N	N	-	DISCHARGE
115	1404307	-	-	Y	-	3D	N	N	-	DISCHARGE
116	1406282	-	-	Y	-	4D	N	N	-	DISCHARGE
117	1406907	-	Y	-	-	5D	N	N	-	DISCHARGE
118	1406967	IVIG	-	-	Y	18D	Y	N	-	DISCHARGE
119	1407693	-	Y	-	-	9D	N	N	-	DISCHARGE
120	1350680	-	Y	-	-	4D	N	N	-	DISCHARGE
121	1409007	-	Y	-	-	6D	N	N	-	DISCHARGE
122	1409624	-	Y	-	-	6D	N	N	-	DISCHARGE
123	1409765	-	Y	-	-	7D	N	N	-	DISCHARGE

Baba Farid University
of Health Sciences
Sadiq Road
Faridkot-151 203 (Pb). INDIA.
Phone : 01639-256232,256236
Tele/Fax : 01639-256235
Email – Examination@bfuhs.ac.in

No. BFUHS/2K21p-TH/ 657 Date 19/01/22

The Principal,
DMC, Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.10.2021 has approved the plan of thesis of **Megha Dadu**, a student of your college for **MD General Medicine** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Clinical profile and outcome of patients presenting with severe thrombocytopenia in a tertiary care hospital.

Supervisor
Dr. Vikas Garg
Prof.,
Dept. of Medicine,
DMCH, Ludhiana.

Co-Supervisor

Dr. Dinesh Gupta
Prof. & Head,
Dept. of Medicine,
DMCH, Ludhiana.

Dr. Bhavna Malhotra
Prof.,
Dept. of Pathology,
DMCH, Ludhiana.

The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

55. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*
56. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

CLINICAL PROFILE AND OUTCOME OF PATIENTS PRESENTING WITH SEVERE THROMBOCYTOPENIA IN A TERTIARY CARE HOSPITAL

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

DOCTOR OF MEDICINE

IN

MEDICINE

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

MARCH 2021

DR MEGHA DADU

DEPARTMENT OF MEDICINE

DAYANAND MEDICAL COLLEGE AND HOSPITAL

LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	CLINICAL PROFILE AND OUTCOME OF PATIENTS PRESENTING WITH SEVERE THROMBOCYTOPENIA IN A TERTIARY CARE HOSPITAL
For the degree of	MEDICINE
Name of the candidate	MEGHA DADU
Supervisor	Dr Vikas Garg, Professor, Department of Medicine, DMC&H Ludhiana
Co-supervisor	Dr Dinesh Gupta, Professor & Head, Department of Medicine, DMC&H Ludhiana Dr Bhavna Malhotra, Professor, Department of Pathology, DMC&H, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Thrombocytopenia is defined as platelet count less than 150 X 10^9 /L. It can further be classified as

Mild-above 70 X 10^9 /L

Moderate - platelet count 20 to 70 X 10^9 /L and

Severe - platelet count less than 20 X 10^9 /L(1)

Severe thrombocytopenia is associated with increased morbidity and mortality, which compels the need for accurate diagnosis and proper treatment. This is a prospective study which aims to describe the various risk factors, clinical presentations, various interventions taken, the response achieved from the interventions and also to examine the impact of severe thrombocytopenia on patient outcome.

Aim of Study: 1. To study the clinical profile of patients presenting with severe thrombocytopenia. 2. To study the outcome of patients with severe thrombocytopenia

Materials & methods: A prospective study will be conducted which will include patients presenting with severe thrombocytopenia in Medicine and allied specialties OPDs and wards at Dayanand Medical College and hospital, Ludhiana From 1/03/2021 to 28/02/2022

Clinical Significance: severe thrombocytopenia can be due to various etiologies and is associated with morbidity and mortality. This study will help in understanding the ever changing trends in presentations and correlating those with the outcomes

Keywords: Prospective study, clinical profile, outcome, severe thrombocytopenia.

(Signature)	(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-supervisor	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"CLINICAL PROFILE AND OUTCOME OF PATIENTS PRESENTING WITH SEVERE THROMBOCYTOPENIA IN A TERTIARY CARE HOSPITAL"** exist in the Department of General Surgery, DMC&H, Ludhiana and will be provided to be candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate himself under our supervision and guidance.

Supervisor

Dr Vikas Garg
Professor
Department of Medicine
DMCH, Ludhiana

Co-Supervisor(s)

Dr. Dinesh Gupta
Professor & Head
Department of Medicine
DMCH, Ludhiana

Dr. Bhavna Malhotra
Professor
Department of Pathology
DMCH, Ludhiana

HEAD OF DEPARTMENT

Dr. Dinesh Gupta
Professor and Head
Department of Medicine
DMCH, Ludhiana

Place: LUDHIANA

Date: 12/3/21

APPROVAL PROFORMA
BY RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Megha Dadu
Department	Medicine
Topic of Thesis	CLINICAL PROFILE AND OUTCOME OF PATIENTS PRESENTING WITH SEVERE THROMBOCYTOPENIA IN A TERTIARY CARE HOSPITAL
Likely date of appearing in PG examination	MAY 2023
Date of enrollment	30 June 2020
Name of Head of Department	Dr. Dinesh Gupta
1) Supervisor **2) Co-Supervisor**	Dr. Vlkas Garg Dr. Dinesh Gupta Dr. Bhavna Malhotra
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Harpreet Kaur (Professor & Head, Pathology)	Mr. R K Sharma (Legal Expert)
Dr. Jaspal Singh (Professor & Head, Surgery)	Dr. Ramit Mahajan (Associate Professor, Gastroenterology)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)	Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)
Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal & Professor & Head, Cardiology)
	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / No

Dr. Sandeep Puri
Chairperson
Research Committee

Approved: Yes / ~~No~~

Prof. Arvind Malhotra
Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	Dr. MEGHA DADU POST GRADUATE STUDENT, DEPARTMENT OF MEDICINE, DAYANAND MEDICAL COLLEGE AND HOSPITAL,LUDHIANA
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL COLLEGE AND HOSPITAL LUDHIANA
3	COURSE OF STUDY AND SUBJECT	DOCTOR OF MEDICINE IN GENERAL MEDICINE
4	DATE OF ADMISSION OF COURSE	30 JUNE 2020
5	TITLE OF THE TOPIC	CLINICAL PROFILE AND OUTCOME OF PATIENTS PRESENTING WITH SEVERE THROMBOCYTOPENIA IN ATERTIARY CARE HOSPITAL

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Platelets are anucleate blood cells produced in the bone marrow by megakaryocytes.

Thrombocytopenia i.e. deficiency of platelets causes coagulopathy which can lead to severe bleeding manifestations resulting in significant morbidity and mortality. It is defined as platelet count less than 150 X 10^9 /L. It can further be classified as

Mild-above 70 X 10^9 /L

Moderate -platelet count 20 to 70 X 10^9 /L

Severe-platelet count less than 20 X 10^9 /L [1]

The reference range for human platelet count determined by automated particle counters is usually considered to be between 150-450 X 10^9 /l. However the range of platelets count that is consistent with normal coagulation is much wider, between 50-600 X 10^9 /l [2]

Thrombocytopenia is variably attributed to many factors such as decreased platelet production, increased platelet destruction, or abnormal splenic pooling. Thrombocytopenia is a recognized side effect of treatment with a wide range of medications .Certain agents particularly those used for chemotherapy tend to cause bone marrow suppression and produce pancytopenia. Heparin induced thrombocytopenia, 'quinine' type immune thrombocytopenia and thrombocytopenia induced by glycoprotein IIb –IIIa inhibitors are most likely responsible for the drop in platelet count in patients seen clinically.

The most common hematologic etiologies are immune thrombocytopenic purpura, heparin induced thrombocytopenia, in vitro platelet clumping, myelodysblastic syndrome and thrombotic thrombocytopenic purpura. The most common non hematologic etiologies are liver disease, sepsis and oral medications. [3]

Bleeding manifestations in the form of subconjunctival hemorrhage, epistaxis, gum bleeding, purpura, echymoses, malena can occur due to thrombocytopenia.

Observational data of ITP patient cohorts have suggested that bleeding risk is increased with platelet counts less than 20 or 30 x 10^9/L. [4]

There are various drugs used in patients with decreased platelet counts including steroids, immunoglobulin,anti-D,thrombopoietin receptor agonist, and surgical options like splenectomy is also there.

The optimal threshold platelet count for prophylactic platelet transfusions in patients with hematological malignancy undergoing cytotoxic therapy remains uncertain. Recent studies have suggested that for patients with no additional risk factors, the clinical decision threshold may be reduced from a platelet count of 20 X 10^9/l to 10 X 10^9/l without significantly increasing the risk of hemorrhage.[5]

Severe thrombocytopenia can increase the morbidity and mortality of the patient. The outcome of patient can be affected significantly in presence of it.

Previous studies have identified thrombocytopenia as an independent marker of longer ICU stay and increased overall and ICU mortality.[6]

This study can provide baseline data for future clinical research. Theobjective of this study is to describe the various risk factors, clinical presentations, various interventions taken, the response achieved from the interventions and also to examine the impact of severe thrombocytopenia on patient outcome.

6.2 REVIEW OF LITERATURE

Michael F Buckley et al. assessed the significance of numerical changes in the platelet count based upon a result exceeding the normal intra –individual variation in platelet numbers by taking serial platelet counts from 3,789 subjects and determine the intra individual variation in platelet numbers. The repeatability of the platelet count was calculated as 0.871(males) and).849(female) indicating that the heritability of platelet count is high and the platelet count is predominantly genetically determined. [2]

MD Michael boxer et al. studied the etiologies of thrombocytopenia in the community hospital and found that Thirty-five patients had a primary hematologic cause for their thrombocytopenia. Fifty-one had secondary causes for thrombocytopenia. Eleven patients had no discernible etiology for their thrombocytopenia. The most common hematologic diagnoses were immune thrombocytopenic purpura (n = 12), heparin-induced thrombocytopenia (n = 6), in vitro platelet clumping (n = 4), quite remarkably acute progranulocytic leukemia (n = 4), and thrombotic thrombocytopenia purpura (n = 3). The most common nonhematologic etiologies were liver disease (n = 21), bacterial sepsis (n = 12), and oral medication (n = 7) [3]

Mazza P, Minoia C et al. did a multicenter study in which a total of 124 patients were retrospectively evaluated: 55 (44.3 %) were treated by romiplostim and 69 (55.6 %) by eltrombopag. Mean age, number of young patients (<60 years), time from primary diagnosis of ITP to TPO-RA treatment, and previous lines of therapy were similar in both groups. The overall response rate was 80 % (44/55) for romiplostim and 94.2 % (65/69) for eltrombopag; the duration of response and the time to response were similar (p = NS). The mean duration of response was 30 months for romiplostim and 15 months for eltrombopag, due to later commercialization of eltrombopag. [4]

Crowther MA et al. examine the impact of thrombocytopenia developing in intensive care unit on patient outcome in a well defined cohort of critically ill patients in a medical-surgical ICU.Ofthe 261 enrolled patients,121(46%,95% confidence interval[CI],40%-50%)had

thrombocytopenia(62 on ICU admission and 59 acquired during ICU stay. Patients who developed a platelet count less than 150 x 10^9 /L during their ICU stay had higher ICU and hospital mortality(p=0.03 and 0.005 respectively),required longer mechanical ventilation(p=0.005) and red blood cell transfusions(p=0.004)than patients who did not develop thrombocytopenia. [6]

Segal hc et al. found variation in platelet counting between different analysers and even the same type of analyser at different sites. Optical methods for platelet counting on the XE 2100, Advia 120, Cell-Dyn 4000 and H3* were not superior to impedance methods on the XE 2100, LH750 and Pentraanalysers. All analysers except one overestimated the platelet count, which would result in under transfusion of platelets. [5]

Chang H et al. studied that Among 665 patients with thrombocytopenia, the diagnosis of ITP was made in 375. Two hundred and fifty-seven patients (51 males, median age 45.5) received treatment. Response to steroids was evaluable for 184 patients. Complete response (CR) was achieved in 120 (65.2%) and partial response in 43 (23.3%). In 21 (11.4%) patients, ITP was refractory to steroids. They concluded that Steroids are effective frontline treatment for ITP, but relapse is common. Both azathioprine and splenectomy are effective treatment after steroid failure. Rituximab appears to a reasonable second-line treatment option in our limited experience. [7]

SerebruanyVL et al didthe 10-day treatment with eltrombopagwhich increased platelet count up to 1.5 times. The drug is usually tolerated well and serious adverse events and discontinuation are rare. There is an unmet need for this agent, and probably eltrombopag is indeed the ‘‘drug of choice’’ for some limited categories of patients; however, its long-term safety profile is unknown. [8]

Williamson David R. Et al. described the prevalence, incidence,and risk factors for thrombocytopenia in the intensive care unit(ICU) and evaluated the impact of thrombocytopenia on mortality with further comparisons amongst major diagnostic categories. The study included 20,696 patients .Prevalent and incident thrombocytopenia occurred in

13.3% and 7.8% of patients, respectively, with associated mortality rates of 14.3% and 24.7% respectively, compared with 10.2% in the group with normal platelet count($p<0.001$). [9]

Michael boxer et al. concluded that most common hematological diagnoses were immune thrombocytopenic outputs, heparin induced thrombocytopenia, in vitriol; plateletclumping, quite remarkably acute progranulocytic leukemia and thrombotic thrombocytopenic purpura and the most common non hematological etiologies were liver disease, bacterial sepsis and oral medications. [10]

Strauss Richard et al. concluded that thrombocytopenia patients have a higher prevalence of bleeding and greater transfusions requirements. They also concluded that a drop in platelet counts of more than 30 %, but not thrombocytopenia per se,is independently associated with intensive care unit death[11]

Arnold dm et al. studied bleeding complications in immune thrombocytopenia and found that Severe bleeding is distinctly uncommon when the platelet count is >30 X 10^9 L and usually only occurs when the platelet count falls <10 X 10^9)/L and. [12]

Eric M fountain et al. did a retrospective study of all patients admitted to a general medical ward from January 1st, 2014 to December 31st, 2014 with hospital acquired thrombocytopenia. In their study, the patients with moderate thrombocytopenia (platelet less than 100 X 10^9 /l and /or a >50%relative decline, were compared to those with less severe thrombocytopenia. They concluded that severe sepsis and antibiotic use were both associated with moderate thrombocytopenia, including frequency of HIT testing and red blood cell transfusions.Outcome metrics including transfer to an intensive care unit,death during admission and length of stay were all associated with moderate thrombocytopenia.[13]

K peter salonen et al. studied In 83 treatment programs; the mean recovery at the one-hour posttransfusion time point was 8.6 X 10^9platelets/1 with fresh platelets and 5.9 X 10^9platelets/1 with stored platelets. Significantly better recovery was achieved with freshly prepared platelet over the total of platelet concentrates stored for up to

96 hours; however, if the recoveries in different patient groups given stored platelets were considered separately in terms of storage times of up to 48 h or 48 - 96 h, the good recovery with fresh platelets was significantly better only[14]

Barbara j bain et al. studied the ethnic and sex differences in the total and differential white cell count and platelet count and concluded that Africans and Afrocaribbeans had lower total white cell, neutrophil and platelet counts than Caucasians and counts were lower in Africans than in Afrocaribbeans. Women had higher neutrophil and platelet counts than men in all ethnic groups. [15]

Jaanalatvala et al. concluded in their study that abnormal platelet and leukocyte levels were common, especially in the anemic alcoholics. Both increased mean cell volume of erythrocytes (macrocytosis; 67 vs. 18%; $p < 0.0001$) and mean cell hemoglobin (63 vs. 22%; $p < 0.0001$) were more frequent in the alcoholics than in the nonalcoholic. Reticulocytosis (37%), thrombocytopenia (41%), and combined cytopenias (34-38%) were also common findings in the alcoholic patients. The blood smears from such patients typically showed round macrocytes, stomatocytes, and knizocytes. Bone marrow aspirates revealed vacuolization of pronormoblasts in 24% of the alcoholic patients. [16]

Rodeghiero F et al. defines the term response asplatelet count more than or equal to 30 X 10^9 /l and at least 2 fold increase in the baseline value and absence of bleeding. Complete response is defined as platelet count more than or equal to 100 X 10^9 /l and absence of bleeding and no response is defined as platelet count less than 30 X 10^9 /l or less than 2 fold increase of baseline platelet count or bleeding. [17]

Neunert c et al in their study gave guidelines When patients with ITP and thrombocytopenia require a rapid rise in their platelet count to achieve adequate hemostasis, physicians are limited in their treatment options considering that the standard treatments for ITP take many hours or days to have an effect. As discussed in section 4.3, IVIg is

proven to have the most rapid onset of action (grade 2B) and should be considered along with corticosteroids (grade 2B) with the aim of increasing the platelet count.[18]

Zarychansky et al concluded in their study that The assessment of thrombocytopenia begins by looking back to the patient's medical history and presenting illness. This past information, combined with careful observation of the platelet trajectory in the context of the patient's clinical course, offers clues to the diagnosis and prognosis. Management is primarily directed at the underlying disorder and transfusion of platelets to prevent or treat clinical bleeding. [19]

Y.K MAT et al. suggest that BME is not helpful in the diagnosis of isolated thrombocytopenia or suspected ITP in adult patients at presentation, provided that a thorough clinical history and physical examination are undertaken and that the blood count and peripheral blood smear show no abnormalities apart from the thrombocytopenia.[20]

6.3 AIMS AND OBJECTIVES OF THE STUDY

- To study the clinical profile of patients presenting with severe thrombocytopenia
- To study the outcome of patients presenting with severe thrombocytopenia

7.0 MATERIALS AND METHODS

A prospective study will be conducted in patients presenting with severe thrombocytopenia in Medicine and allied specialities OPDs and admitted at Dayanand Medical College and hospital, Ludhiana From 1/03/2021 to 28/02/2022. The patients will be followed up prospectively during their hospital stay and further followed up on OPD basis for 6 weeks or till the response is achieved (whichever is early). The demographic data, including patient age, sex, and diagnosis at the time of admission, clinical and laboratory findings will be collected and recorded in a proforma for analysis and interpretation of data.

Data and observations describing possible complications of thrombocytopenia, such as bleeding manifestations (Multiple bleeding events at the same location will be counted only once for each patient), shock will be collected and recorded in the proforma.

The data of various types of intervention including number and types of transfusions (random donor platelets/single donor platelets, fresh frozen plasma, and packed red blood cells), drugs like steroids, thrombopoietin agonists,IVIG,Anti -D given to the patient will also be recorded. The response of intervention will be measured in terms of complete response, partial response and no response. Response is defined as platelet count more than or equal to 30 X 10^9 /l and at least 2 fold increase in the baseline value and absence of bleeding. Complete response is defined as platelet count more than or equal to 100 X 10^9 /l and absence of bleeding and no response is defined as platelet count less than 30 X 10^9 /l or less than 2 fold increase of baseline platelet count or bleeding.

The outcome of the patients in terms of duration of hospital stay, need for ICU care, need for mechanical ventilation will be observed and recorded.

7.1 SOURCE OF DATA

Patients with severe thrombocytopenia attending Medicine and Allied specialties, admitted in Medicine and Allied specialties wards at Dayanand Medical College and hospital,Ludhiana from 1/03/2021 to 28/02/2022

7.2 METHOD OF COLLECTION OF DATA

Cases will be selected on the basis criteria set in inclusion and exclusion criteria for the study

Inclusion Criterion:

1) Patient with severe thrombocytopenia (less than 20 x 10^9 /L)
2) Patient age more than or equal to 18 years

Exclusion Criterion:

1) Patients with mild and moderate thrombocytopenia
2) Patients not willing to take part in the study
3) Age<18 years.
4)patients diagnosed with any malignancy

Outcome:

The objective of this study is to describe the various risk factors, clinical presentations, various interventions taken, the response achieved from the interventions and also to examine the impact of severe thrombocytopenia on patient outcome

Statistical Analysis:

Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

Yes,appropriate investigations will be done whenever required.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Yes

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PROFORMA

Sr. No

NAME	
AGE	
SEX	
MRD NO.	
DATE OF ADMISSION	
WARD	
PHONE NO.	
ADDRESS	

DIAGNOSIS AT THE TIME OF PRESENTATION-

ALCOHOLIC HISTORY-YES/NO

IF YES-

DURATION-

AMOUNT-

TYPE-

H/O TREATMENT-

(CHEMOTHERAPY, HEPARIN.ABCIXIMAB, QUININE, SULFONAMIES, OTHERS)- YES /NO

IF YES THEN-

DRUG-

Signature of Candidate **Signature of Supervisor**

DATE -

DURATION OF DRUG GIVEN-

DATE OF DEVELOPMENT OF THROMBOCYTOPENIA-

FAMILY HISTORY-

RECENT TRANSFUSION HISTORY-

COMORBIDITIES- IF YES –DURATION OF THE COMORBIDITY

TYPE 2 DM

HYPERTENSION

EPILEPSY

BRONCHIAL ASTHMA

COAD

CAD

CVA

CKD

THYROID DISEASE

ANY OTHER

Signature of Candidate **Signature of Supervisor**

SYMPTOMS/SIGNS

SYMPTOMS	
FEVER	
MALAISE/FATIGUE/GENERALISED BODY WEAKNESS	
NAUSEA	
VOMITING	
DIARRHOEA	
CONSTIPATION	
BREATHLESSNSS	
BLEEDING MANIFESTATIONS- SUBCONJUNCTIVAL HEMORRHAGE EPISTAXIS GUM BLEEDING ECHYMOSES MALENA HEMATURIA MENORRHAGIA	
WEIGHT LOSS	
ABDOMINAL PAIN	
ANY OTHER SYMPTOMS	
SIGNS	
RED SPOTS	
RASHES	
HEPATOMEGALY	
SPLENOMEGALY	
LYMPHADENOPATHY	
ANY OTHER SIGNS	

Signature of Candidate Signature of Supervisor

LAB PARAMETERS

CBC

TLC											
NEUTROPHILS											
MONOCYTES											
EOSINOPHILS											
BASOPHILS											
HB											
PCV											
MCV											
MCHC											
RDW											
PLATELET											

TLC											
NEUTROPHILS											
MONOCYTES											
EOSINOPHILS											
BASOPHILS											
HB											
PCV											
MCV											
MCHC											
RDW											
PLATELET											

LFT /RFT

BIL (t)											
BIL(d)											
SGOT											
SGPT											
ALP											

Signature of Candidate **Signature of Supervisor**

ALBUMIN											
TOTAL PROTEINS											
UREA											
CREATININE											
Na											
K											
Cl											

PBF	
BMA AND BIOPSY- (IF DONE) HYPERCELLULAR NORMOCELLULAR HYPOCELLULAR MEGAKARYOCYTES- OTHER-	
ANA	
APLA	
VIRAL MARKERS	
TROPICAL FEVER	
D DIMER	
PROTHROMBIN TIME	
FIBRINOGEN	
ANY OTHER	

Signature of Candidate

Signature of Supervisor

RADIOLOGICAL INVESTIGATION

USG WHOLE ABDOMEN	
CT ABDOMEN	
ANY OTHER	

INTERVENTION

RESPONSE

RDP		COMPLETE	
SDP		PARTIAL	
STEROIDS DRUG- DOSE- DURATION-		NO RESPONSE	
TPO AGONIST DRUG USED- STARTED ON- DOSE- DURATION-			
OTHER INTERVENTION (IVIG/SPLENECTOMY)			

Signature of Candidate

Signature of Supervisor

OUTCOMES

LENGTH OF HOSPITAL STAY	
NEED FOR ICU CARE	
NEED FOR INTUBATION – DATE DURATION	
DISCHARGE /DAMA/MORTALITY	

Signature of Candidate

Signature of Supervisor

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o.....
.....am willing to participate in the study **"CLINICAL PROFILE AND OUTCOME OF PATIENTS PRESENTING WITH SEVERE THROMBOCYTOPENIA IN A TERTIARY CARE HOSPITAL"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

.....
Signature of the Witness

.....
Signature of the patient

Name:

Signature of Candidate

Signature of Supervisor

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य "**CLINICAL PROFILE AND OUTCOME OF PATIENTS PRESENTING WITH SEVERE THROMBOCYTOPENIA IN A TERTIARY CARE HOSPITAL**" में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____

गवाह के हस्ताक्षर

नाम:

_____

रोगी के हस्ताक्षर/अंगूठे का निशान

नाम:

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अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"CLINICAL PROFILE AND OUTCOME OF PATIENTS PRESENTING WITH SEVERE THROMBOCYTOPENIA IN A TERTIARY CARE HOSPITAL" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

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ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

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ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ